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ENCYCLOPAEDIA
OF
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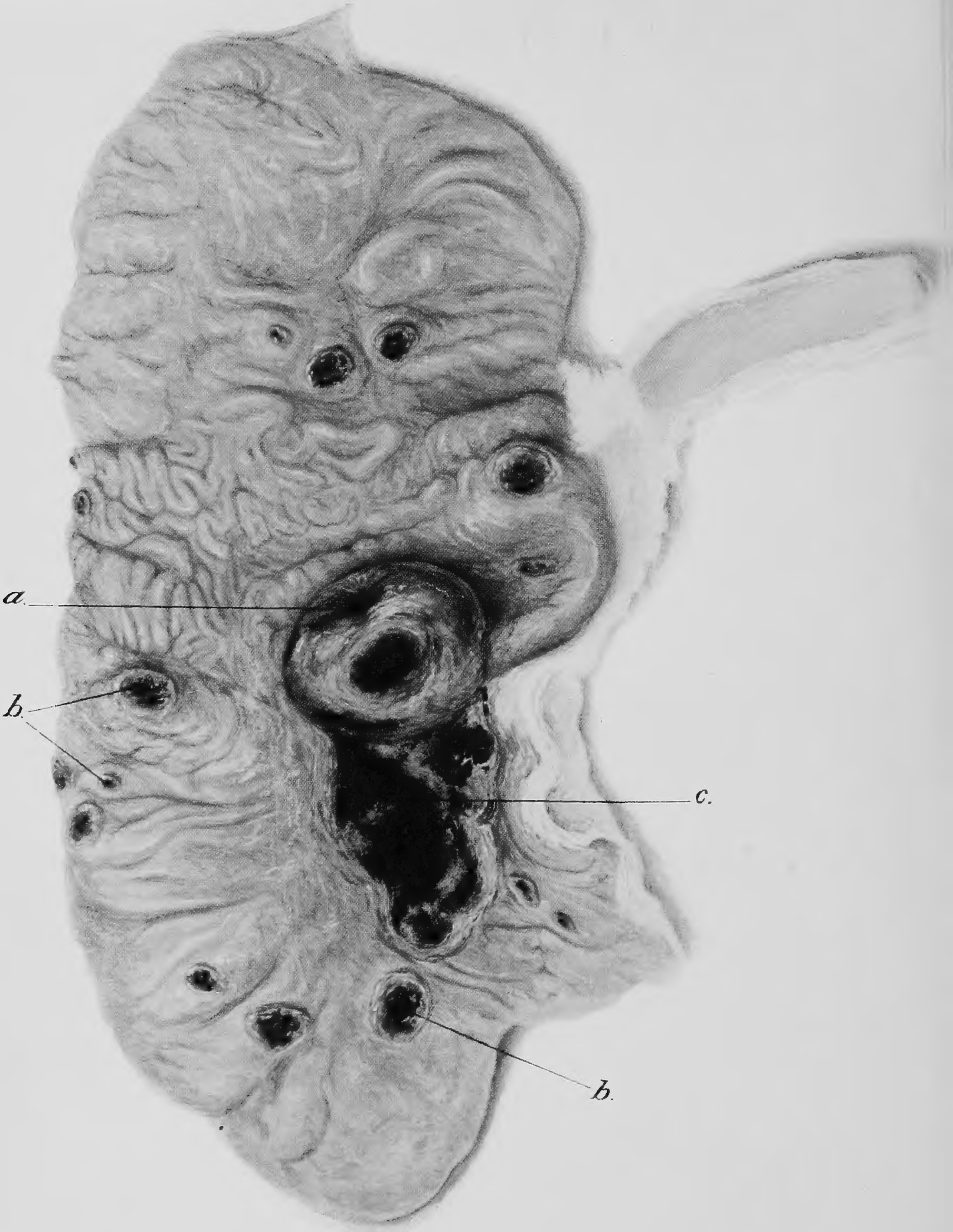

SWINE FEVER.

Mucous surface of caecum and part of colon from pig dead of swine fever. The ileo-caecal valve is swollen and ulcerated. *a*, ileo-caecal valve; *b*, "button" ulcers; *c*, large ulcer resulting from coalescence of several "button" ulcers. (*Dorset.*)

ENCYCLOPAEDIA
OF
VETERINARY MEDICINE SURGERY & OBSTETRICS

EDITED BY

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IN TWO VOLUMES

VOL. I

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PREFACE

THE great strides that have been made in Veterinary Medicine and Surgery in recent years have rendered it very difficult for the general veterinary practitioner to keep abreast of the times. Moreover, veterinary students have had great difficulty in obtaining up-to-date books dealing with the practical side of their studies in a concise manner. The medical profession is daily realising more and more fully the importance of diseases of animals in relation to diseases of man, but there has been difficulty in obtaining a book to which it could turn for information concerning animal diseases in a succinct form. The object of this work is to supply these needs, and to keep it to such size and scope as to make it easy to handle and simple of reference. With a few exceptions there has been no attempt to go into pathological details beyond those necessary to afford a basis for understanding the causes, course, and diagnosis of the affection. It is hoped that nothing of real importance has been omitted, and that busy practitioners and students of veterinary medicine and surgery will find it of value and assistance. The numerous illustrations should also be found helpful.

There is also another great section of the community, namely, the owners of valuable animals, who may find the work of service and interest in enabling them to realise the difficulties of the veterinary practitioner. The Editor has a firm belief in the education of animal owners in such matters, and he is of opinion that educated men, reading articles such as are provided in this book, make by far the most satisfactory clients. They realise the necessity for careful nursing and for attention to detail in following the instructions given by the practitioner, and are less likely to rely with blind faith upon some quack nostrum guaranteed to cure all ills, and purchased at small cost in the market-place or elsewhere from the plausible medicine-vendor.

It has been the Editor's endeavour to select experts to contribute articles in clear and lucid language on their own subjects. He is grateful to each and all of them for the very valuable assistance they have rendered. It is not possible to single them out for individual mention here, with one exception, namely, Professor James Macqueen, who practically edited the Surgical Section, and without whose generous co-operation the book, long delayed as it has been in its preparation, could not have been completed for some time to come.

Some difficulty was experienced as to the best arrangement to adopt, and after much thought the systemic arrangement rather than the alphabetical has been adopted. It is hoped that this will be found more generally convenient. It can be seen at a glance in the Table of Contents at the commencement of Volume I., while an ample Index at the end of each volume will facilitate reference to any particular subject.

In submitting the work, the Editor would invite that any defects may be pointed out to him, so that they may be remedied in the event of another edition being called for.

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VETERINARY MEDICINE

INTRODUCTION

VETERINARY Medicine concerns itself with the ailments of animals which do not as a rule call for manipulatory interference. The *theory of medicine* enumerates the principles on which disease acts and on which remedies are applied, while the *practice of medicine* consists of the adaptation of those principles to the treatment of disease and the relief of pain. Where medicine is practised without being based on those principles it is called *Empirical Medicine*, and is closely allied to " quackery."

We have already used the word disease, and it is therefore necessary to define it. It is, however, practically impossible to do so except in terms of health, since " *Disease* is any departure from health." *Health*, on the other hand, is " that condition of structure and function which, on the examination of the largest available number of individuals, is found to be the common or prevailing one."

There are certain conditions which are essential to the maintenance of health. They may be summed up under three headings, namely: (1) Proper surroundings; (2) proper food; (3) proper vital activity of the whole organism.

The practice of medicine is applied both to prevent and to remedy any change of the animal body due to disturbance of any of these essential conditions of health.

Preventive medicine or *prophylactic medicine* (*prophylaxis*) is that branch which attempts to maintain the balance of health when disease is imminent. It is thus closely allied to hygiene, which treats of the preservation of health and the prevention of disease but not where the latter is imminent. Parke's definition of hygiene is perhaps the best and most comprehensive yet formulated, as follows :—

" Hygiene aims at rendering growth more perfect, decay less rapid, life more vigorous, and death more remote."

Remedial or *curative medicine* is applied to the restoration of health.

Conditions or Signs of Health.—A healthy animal should eat, drink, and sleep well and regularly, and should be bright in manner. The posture should be normal and easy, disease being indicated by irregularities such as resting limbs, outstretched nose, pendulous ears or lips, inappetence, etc. Change of habits often indicates disease. The various functions must also be normal, such as the pulse, respiration, temperature both internal and external, and excretions. Rumination in the case of ruminants should be regular, and the same applies to lactation in milk-yielding females.

Disease. — Disease may be classified in various ways. It may be (*a*) *Hereditary*, (*b*) *Acquired*. In the broad sense any disease handed in any way from parent to progeny is called *hereditary*. In the more restricted sense a disease is only regarded as hereditary if it is transmitted directly through the ovum or sperm. A *congenital disease* is one which is appreciable at birth, and is consequently popularly regarded as one form of hereditary disease. All diseases which are not hereditary or congenital are *acquired*.

Another classification is the division into (*a*) *General* disease, (*b*) *Local* disease.

A *general disease* is one in which systemic disturbance usually precedes local change or is early associated with it. A *local disease* is one in which local change is primary and remains predominant throughout. In such a case it is implied that the diseased condition first involves a single organ or tissue. The disease may, however, be very extensive and yet properly designated a " local disease," as in the case of some skin affections.

Sometimes disease is classified into (*a*) *Structural*, (*b*) *Functional* disease.

A *structural disease* is one in which there is appreciable alteration in structure. A *functional disease* is one in which there is alteration in function but in which no alteration of structure is appreciable It is doubtful whether a truly functional disease really exists, inasmuch as although there may be no gross changes of structure such as are observable by the naked eye, yet it is more than probable that if a thorough and exhaustive microscopic examination were made, some slight structural alteration as of nerve cells or terminals might be discovered.

A *specific disease* is one produced by a definite organism and in which definite changes are produced in more or less constant succession. The term "specific disease" usually implies a contagious disease, or one which can be transmitted by some means or other from animal to animal. It often happens, therefore, that when a specific disease occurs, cases are multiple. On the other hand, when a disease occurs and attacks an individual, and shows no tendency to spread to other animals, it is called a *sporadic disease.*

ETIOLOGY OR CAUSATION OF DISEASE

The causes of disease may be divided into two classes, or may be studied under two headings, namely: (1) Predisposing causes; (2) exciting causes. The predisposing causes of disease are those which appear to render the animal body less resistant to the exciting causes. They consist of circumstances which influence the body in some unfavourable way and yet of themselves do not actually set up the disease.

Predisposing Causes of Disease.—1. *Age.* Both young and old animals are predisposed to disease by virtue of their age which animals in the prime of life rarely contract. In the case of young animals we have such examples as strangles in horses; distemper in dogs; blackquarter in calves; rickets in any species of animals. In old age, on the other hand, one constantly meets with disease associated with debility of tissue, such as weak circulation, indigestion, and the presence of tumours. Such affections are comparatively rarely met with in young animals. Youth predisposes to bacterial diseases, while advanced age predisposes to diseases associated with debility of tissue.

2. *Sex.* The difference in the anatomical structures of the sexes obviously gives rise to certain affections of organs in one which cannot arise in the other, such as metritis in the female and orchitis in the male.

3. *Temperament* is regarded by some as a predisposing cause of disease; for example, the so-called *lymphatic temperament* which is associated with the general dulness of disposition and with extra development of subcutaneous tissues, œdema of the extremities, and which, however, is probably due to a weak circulation and in such cases would be more correctly regarded as a symptom rather than a predisposing cause of disease. *Nervous temperament* also exists and is generally indicated by the ease with which an animal may be frightened. It is sometimes quite a serious condition. It is doubtful, however, whether it should be regarded as a predisposing cause of disease or rather as a symptom of some defect in the higher nervous system.

4. *Colour.* Colour in some cases undoubtedly is a predisposing factor, and this is particularly exemplified by the occurrence of melanotic tumours in grey horses. It is curious, however, that when melanotic tumours occur in cattle it is most frequently in the reds and not in the greys, while in dogs, in the writer's experience, they occur far more frequently in brown-coated animals, such as Irish terriers.

5. *Anatomical conformations* sometimes predispose to disease; for example, too sloping pasterns are likely to be associated with strained tendons, while too upright pasterns predispose to ringbones.

6. *Previous Disease.* In a general way non-specific diseases predispose to second attacks, while specific or bacterial diseases usually indispose to second attacks by virtue of the fact that they so frequently leave behind them some degree of immunity or tolerance. There are, however, exceptions to this latter fact, such as sporadic pneumonia, which, although due to specific causes, usually predisposes to a recurrence; and, moreover, no one would suggest that one attack of boils or acne due to micrococci leaves behind it any resistance to subsequent attacks. Rather does one unfortunately know that a boil is too frequently a forerunner of successive boils.

7. *Sudden alterations of temperature* may and undoubtedly do predispose to some diseases. The sudden exposure to cold is popularly supposed to be a cause of disease, but it is very difficult to understand how cold, unless in very extreme cases, such as frost-bite, can operate as an exciting cause of disease. It is far more probable that sudden changes of temperature may and do somewhat impair the normal powers of resistance to disease, and so by reducing vitality enable the exciting cause, such as bacteria, to operate. In most cases where a sensation of coldness is followed by catarrh, the rigor or shivering fit is in reality the first stage of the affection, and is therefore an early symptom of approaching fever rather than the cause of it.

8. *Diathesis* or *Idiosyncrasy.* This is the peculiar state of the whole or part of the body which renders it especially favourable to the occurrence of some disease, or conversely endows the individual with resistance against it. The condition is very vague, but it is the only explanation that can be offered in cases where one animal alone may become affected with a disease though living with others under exactly similar conditions.

9. *Heredity* may also be regarded as a predisposing cause of various diseases, as in the case of roaring, navicular disease, etc., in horses.

10. *Climate.* The influence of climate must be acknowledged, particularly because under certain conditions of climate exciting causes of disease are more prevalent. This is especially shown in tropical regions, where the carrier of infection often consists of various insects which only thrive in tropical zones. There are numerous examples of this phenomenon, such as the malarial districts for human beings and tsetse-fly zones in Africa.

Exciting causes of disease are those which are directly associated with the lesions or symptoms and are actually responsible for setting up of the diseased condition. They may be *intrinsic* or *endopathic* in origin, in which case they rise within the animal itself and are entirely independent of external agency, as in the case of debility of tissue. On the other hand, exciting causes may be *extrinsic* or *exopathic* in that they may be quite external in origin so far as the animal economy is concerned.

Exciting causes may also be classified according to their nature into (1) *Mechanical*, such as may result from actual violence, or consisting of some stricture or an obstruction as in the case of calculus or stone. (2) *Chemical*, such as may arise from the accidental or malicious administration or application of irritant or corrosive agents such as acids, alkalies, etc. (3) They may be *thermal*, such as in the case of intense cold causing frostbite, or the other extreme setting up such conditions as heat stroke, burns, or scalds. (4) Causes of diseases may be *dietetic* in origin. Proper food and water must be regarded as essential conditions of health. Food can become an active cause of disease when it is defective either in quantity or in quality, and the same applies to water. Irregular feeding is a frequent cause of colic in horses. (5) Exciting causes of disease may be *vital* or organismal, consisting of parasites of animal or vegetable origin. In medical diseases this is by far the most important class of exciting causes and includes such widely divergent examples as Insects, Worms, Protozoa, Fungi, and Bacteria.

SEMIOLOGY; SYMPTOMATOLOGY; THE STUDY OF SYMPTOMS

A *symptom* may be defined as an appreciable manifestation of a diseased condition. Such manifestation may be somewhat vague, and only serve to indicate that there is "some departure from health," and it does not necessarily indicate the exact nature of the disease or that departure from health. Where a symptom is somewhat more definite, and is more or less directly indicative of the seat of the disease, then it may be called a "sign." It is by the observation and intelligent interpretation of symptoms that one is enabled to ascertain the nature of the disease which gives rise to those symptoms, or, in other words, form a *diagnosis*.

Subjective and *Objective symptoms* are recognized. *Subjective* symptoms are those which are experienced by the patient and voluntarily expressed by him to a physician. It is obvious, therefore, that truly subjective symptoms are not met with in veterinary practice except in fables, such as the classical instance of the lion with the thorn in his foot. On the other hand, the owner or attendant of the patient usually volunteers his impressions and opinions of the symptoms, and such may be regarded by us as taking the place of subjective symptoms. Obviously they are not so reliable as subjective symptoms in the case of a human patient, and the veterinary surgeon has therefore to attach far greater importance to the symptoms he may discover for himself on his examination of the patient These are called *objective* symptoms.

Symptoms which arise early when the balance of health is upset, and are therefore a warning of the approach of some disease, are called *premonitory* or *precursory symptoms*, or *prodromata*. Such symptoms are of themselves not especially indicative of the disease developing; for example, in horses with numerous diseases one of the first things to be observed is loss of appetite, yet this does not in any way indicate that it is the alimentary tract that is affected any more than the respiratory tract.

Direct or *idiopathic symptoms* are those which point straight to the seat of the disease and are immediately associated with the diseased part. *Indirect symptoms* are not directly associated with the diseased part, but they may arise owing to the disturbance of functions of the diseased area. Such symptoms are commonly called *secondary* or *sympathetic symptoms*. *Remittent* or *periodical symptoms* are those which entirely disappear and reappear after longer or shorter intervals. *Diagnostic* or *pathognomonic symptoms* are those which point directly or definitely to one particular disease and afford a certain basis for diagnosis. Diagnostic symptoms are comparatively rare, but may consist of the discovery of the specific organism in any bacterial affection. For example, in the case of anthrax, the demonstration of the *Bacillus anthracis* enables one to form an absolutely definite diagnosis. In most affections it is by a combination of symptoms that the diagnosis is made, and in such cases no one of those symptoms can be regarded as diagnostic. *Negative symptoms* are sometimes spoken of, and although the term appears at first to be paradoxical inasmuch as it implies the absence of symptoms, yet it may with advantage be retained, since the failure to react to the tuberculin test would legitimately be regarded as a negative symptom of tuberculosis, while, on the other hand, a definite reaction to tuberculin would in contradistinction be regarded as a positive symptom.

Prognostic symptoms are those which afford a basis for determining the duration, course, or termination of a disease (*Prognosis*).

Complications are phenomena of a different nature from those caused by the primary disease which may be developed during the course of that disease, such as bed-sores in cases involving prolonged recumbency. Any defective condition which is left after recovery or apparent recovery, or which remains as a change due to the disease, is called a *sequela*. Paralysis is a frequent sequel to apoplexy.

Diagnosis.—Diagnosis consists in determining the nature of the affection from which an animal is suffering. It may be arrived at in several ways. The first step is to assure oneself that there is some departure from health. Although in our patients malingering is not likely to occur very frequently, yet, on the other hand, one frequently has to contend with an owner or attendant whose imagination leads him to believe and state in no measured terms that his animal is ill. This is particularly the case with hysterical owners of pet dogs. The full statement of the owner or attendant should therefore be first elicited, beginning as far as possible with the voluntary history and supplemented with judicious questions—first, as to how long the animal has been noticed ill, the nature of the symptoms which have given rise in the owner's mind to the opinion that the animal is ill, and if possible the circumstances which led up to the illness. Inquiries should also be made as to the appetite, the nature, character, and quantity of food and water which has been partaken of, the character of the excretions (fæces, urine, etc.), the number of animals affected, and possible contagion. One next proceeds to examine the patient, to ascertain the presence or absence of symptoms which support or negative the conclusions arrived at by the owner, and also to discover various other symptoms which may be exhibited, and which will form the basis upon which diagnosis can be made. This examination should first consist of inspection, which should be carried out before the patient is disturbed, noting particularly the posture, character, and number of respirations and the general attitude. One should next proceed to take the pulse, to examine the mucous membranes, noting the age, to take the temperature both internal and external, to examine excretions and secretions, and then proceed to more intimate examination, as by palpation, percussion, and auscultation. Having completed the examination, diagnosis may be arrived at either by a *positive* or by a *negative process*. The former is based upon positive symptoms, while the latter is based largely upon the absence of symptoms associated with special parts and is called a *process of exclusion*.

Symptoms connected with the Pulse.—The pulse is a wave of blood passing through the arteries as the result of each contraction of the heart. It should be taken by the finger-tips on a medium-sized artery, having a firm background. In this way any variation can readily be detected. It should always be firm and regular, though frequently in dogs of excitable temperament it is intermittent, even in health. In the horse the pulse should beat from 36 to 40 to the minute, and is best taken either at the submaxillary artery under the jaw, or at the radial artery inside the elbow. In cattle the average rate is about 50 beats per minute, and it may be taken at the submaxillary artery just outside the lower jaw, the radial artery, the coccygeal artery under the middle of the tail, or the digital artery, which is just above and between the false digits. This latter situation, however, is only serviceable when the animal is in a recumbent position. In the sheep and the pig the pulse is somewhat variable, but averages between 70 and 80 beats per minute. It is best taken at the femoral artery inside the thigh. In the dog and cat the pulse varies according to the breed, and ranges between 80 and 120 beats per minute, and is best taken at the femoral artery. Variations in the pulse beyond certain limits are indications of some departure from health.

When the pulse beats more often than normal in a given time, it is called a *frequent pulse*; when the converse, it is known as an *infrequent pulse*.

A *slow pulse* is one which takes a longer time than normal in passing through its phases. It gives the impression of being slow in passing under the finger. On the other hand, when the individual pulse-beat passes quickly under the finger it is known as a *quick pulse*.

A *hard pulse* and a *soft pulse* are also recognized. These conditions depend upon the degree of contraction or relaxation of the muscle in the arterial wall, and as this in turn depends upon the condition of the nervous system, it is an indication of the general tone of that system.

A *large pulse* and a *small pulse* must also be recognized, and distinguished from the previous category, since they depend entirely upon the degree of fulness of the vessel examined; for example, in certain affections such as congestion of internal organs a considerable proportion of the blood is drawn off to those organs, leaving a smaller amount to pass through the superficial vessels. In such a case a *small pulse* is the result. In many fevers, on the other hand, larger quantities of blood are driven to the body surface for the cooling effect of the atmosphere, and consequently the pulse, as appreciated in a superficial artery, is rendered large. Combinations of the above conditions may also occur; for example, the so-called *wiry pulse* may be met with, and this is a small hard pulse.

Any extra pause between the pulse-beats is called an *intermittence,* and the "intermittence" may be either *regular* or *irregular,* both in its duration and in its occurrence. An *irregularly intermittent* pulse is often a very serious condition, since the pause may sometimes be too protracted and the heart may become over-distended to such a degree as to be unable to resume its function. In some diseased conditions the pulse gradually gets smaller and smaller, until it becomes almost or quite imperceptible. Such a pulse is called a *running down pulse* and may be met with in excessive hæmorrhage either internal or external, or in cases of intense congestion of the more vascular internal organs.

The *jugular pulse* is a wave of blood which may be seen passing up the jugular vein in the region of the neck. This is due to some obstruction to the flow of blood through the right side of the heart. Its importance as an indication of disease varies according to the cause of the obstruction. In some instances it is actually due to heart disease, such as incompetence of the tricuspid valve between the right auricle and ventricle. In other cases the obstruction is due to some disease of the lungs; while in other cases it is of no significance, and may be observed when an animal is in the recumbent position.

Respiration.—In health the chest wall and the abdominal muscles should both take part in the respiratory effort, which should be regular and easy, and free from any suggestion of jerkiness. The true character is most easily observed by watching the flank carefully while the animal is at rest before he is in any way disturbed. The average number of respirations per minute is as follows:—

In the horse from 8 to 12. In the ox from 12 to 16. In the sheep in winter, from 15 to 20; while in the summer respirations may be very hurried and exceed 100. In the pig, dog, and cat there is a wide range, depending partly on age, breed, and excitability, and it ranges from 10 to 20 per minute when at rest.

There is an approximate ratio in health of one respiration to four pulse-beats. In diseased conditions this ratio is upset, and respirations may even exceed the number of pulse-beats.

Where there is an excessive number of respirations in a given time, this condition is called *hyperpnœa.* Any difficulty of respiration is evidenced by exaggerated movements of the chest wall and flanks. Such a condition is called *dyspnœa.* The difficulty may be in inspiration, and it is then called "inspiratory dyspnœa." The converse is called "expiratory dyspnœa." The former is indicated by heaving of the chest wall, sudden contractions of the diaphragm, causing a sudden flopping movement of the abdominal wall or flank. At the same time the head is extended, and the nostrils dilated to facilitate inspiration. In "expiratory dyspnœa" the contractions of the flanks are exaggerated or spasmodic.

In some painful affections of the thorax the chest wall is fixed and the abdominal wall is thrown into excessive play. This condition is called *abdominal respiration.* On the other hand, in some affections of the diaphragm and abdominal cavity, the abdominal muscles are more or less fixed, and the movement of the chest walls becomes exaggerated. This is called *thoracic respiration.* Hence "thoracic respiration" usually indicates some painful condition of the abdomen, while "abdominal respiration" usually indicates some painful condition of the chest, such as pleurisy.

Orthopnœa is the condition in which an animal can only breathe while in the upright position, as in cases where there is a considerable quantity of fluid in the chest. *Deep* and *shallow* respirations are also recognized, the latter usually indicating some degree of debility.

Stertor is the technical term for snoring, and is produced by the vibration of a relaxed pendulous palate. If stertor occurs during any brain affection it must be regarded more or less seriously, since the brain centre controlling this area is in close proximity to certain vital centres.

Grunting is an abnormal sound produced during expiration by forced expiratory effort through a partially closed glottis.

Roaring and *whistling* are abnormal sounds produced during inspiration. In the restricted application of these terms the sounds are produced at the larynx owing to paralysis of some of the muscles (laryngismus paralyticus). The term is also widely applied to include any abnormal sound produced during inspiration, as in some forms of obstruction in the regions of the nose and throat (actinomycosis, etc.).

Wheezing is an audible respiration, resulting from an attack of bronchitis. It is usually associated with some chronic thickening of the bronchial mucous membrane.

Trumpeting or *high-blowing* is an abnormal sound produced by the flapping of the false nostrils. It is not a diseased condition, but it frequently indicates fright or nervousness in horses.

In connection with respiration the odour of the breath is also to be observed. It may be fœtid as the result of ulceration in the mouth or carious teeth, or it may be due to gangrene of the lungs. In some instances the objectionable odour may be symptomatic of indigestion, and is due to the coating of the teeth and the mucous membrane of the mouth, and the breaking down of epithelium by micro-organisms.

Temperature.—*Internal temperature* and *external temperature* are recognized.

The *external temperature* is the temperature of the skin and extremities, and is maintained by the distribution of heat through the circulation. It therefore depends on the circulation in the part of the body examined. It may thus be an indication of the heart's action, and its power to drive the blood through the skin and remote parts of the body. Changes in the external temperature may be brought about by nervous impressions, as in acute pain, and in systemic affections such as those of a febrile nature or those associated with congestion of various internal organs. In acute nervous impressions the walls of the superficial vessels frequently become constricted, producing cold body surface and extremities, and a similar condition occurs in the initial or cold stage of a fever. In fevers, other than the early stages, the external temperature is high and the body surface and extremities feel warmer than normal. In internal congestion and in cases of weak circulation the external temperature will be low owing to deficient amount of blood passing through the superficial vessels. In estimating the external temperature the hand should be passed over the surface of the body and also the ears, muzzle, and limbs of horses, the base of the horns, the muzzle and limbs of oxen, the muzzle of dogs and cats, and the snout of swine. The examination of only a limited area, such as a single leg of a horse, may be very misleading, since that particular leg of an animal having a normal external temperature may be exposed to a small current of air which would reduce the local circulation and produce a cold sensation.

The *internal temperature* is a thermometric measurement of the heat of the blood. It is usually taken in the domesticated animal per rectum, sometimes in the female per vulvam. In the smaller animals the thermometer may be inserted high up between the thighs or in the axilla. The first-named is the best situation. The temperature of health may vary within certain limits in the same animal. The normal temperature is as follows :—

In the horse	from	99·5° F.	to	101° F.	(37·5° C. to 38·5° C.).
In the ox	,,	100° F.	,,	102·5° F.	(38·0° C. ,, 39·0° C.).
In the sheep and goat	,,	101° F.	,,	104° F.	(38·5° C. ,, 40·0° C.).
In the pig (adult)	,,	101° F.	,,	102° F.	(38·5° C. ,, 39·0° C.).
In the pig (young)	,,	102° F.	,,	104° F.	(39·0° C. ,, 40·0° C.).
In the dog	,,	100° F.	,,	102° F.	(38·0° C. ,, 39·0° C.).

In the common fowl the average temperature is 107° F. (42·0° C.).

Care must always be taken to shake the mercury of the clinical thermometer well below the normal temperature of the animal about to be tested, and the instrument should be well inserted into the cavity. If the anus is loose and flaccid, the temperature registered will be slightly lower than that of the blood. The temperature at the vulva is usually a fraction lower than the rectal temperature except at the period of œstrum, when it is usually raised about 0·5° F. In the case of the dog, if the temperature is taken at the axilla, the two surfaces of the skin must be completely around the thermometer, and it is necessary to add 1° F. to the temperature registered.

The normal temperature of any animal is slightly raised after a meal, during rumination and lactation, and in pregnancy of females. Work also raises the temperature somewhat, consequently the normal temperature of the same animal is usually a little higher in the evening than in the morning. It is probably at its highest at about 6 P.M., and its lowest between 4 A.M. and 6 A.M. In young growing animals the temperature is usually a little higher than in animals in the prime of life.

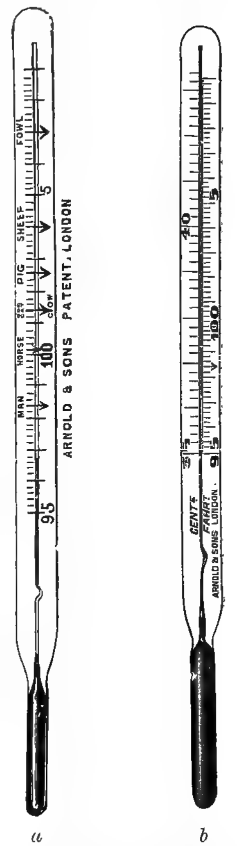

FIG. 1.—Clinical thermometers.

a, Hobday's pattern with the average temperatures of domesticated animals marked off.

b, Pattern showing both Centigrade and Fahrenheit scales.

Temperature above the normal range is usually considered a *febrile* temperature (pyrexia). In cases where the temperature is excessively high the term "hyperpyrexia" is applied. When the temperature is excessively low or sub-normal, it is called a "collapse" temperature. This often indicates the approach of death.

The Visible Mucous Membranes.—These include the conjunctiva, the nasal mucous membrane, the buccal mucous membrane, and the vulva in the female. In health these mucous membranes should be slightly moist and pink. Variations may occur in disease in each of those conditions; thus *dry* and *moist* mucous membranes are recognized. These terms imply respectively that the secretions of the mucous membranes are in abeyance or that they are in excess. The former condition frequently occurs in the early stages of fevers, while the latter occurs in febrile and catarrhal affections, and also in cases of debility of the circulation from various causes. Excessive moisture from mucous membranes is called a *discharge*. When this discharge is thin and watery it is called a *serous* discharge, and is then poor in cellular content. When the discharge is sticky or tenacious it is called a *mucous* discharge, and the viscidity is due to the presence of mucus. A *purulent* discharge is one containing pus, while a catarrhal discharge is one which is rich in epithelial cells (cells shed from the surface of the membrane). When the discharge is blood-tinged it is called a *sanious* or *sanguinolent* discharge. When the discharge from a mucous membrane becomes persistent or chronic it is then termed a *gleet*. In the case of the eyes and the nose, these discharges may be either unilateral, that is from one side only, or bilateral, that is from both sides.

Variations in the *colour* of mucous membranes may be observed, and these are due to the quantity and character of the blood circulating through the blood-vessels of the part. Mucous membranes are said to be *pallid* or *anæmic* when they are paler in colour than normal, owing either to a deficient amount of blood passing through them or to defective amount of hæmoglobin or red colouring-matter in the blood corpuscles. The former condition may be brought about either by hæmorrhage from some part of the body or from congestion of some of the internal vascular organs, such as lungs, spleen, etc. The mucous membranes may also be redder than normal, and are then called *injected*. This is an indication of an increased amount of blood passing through them; and if all the visible mucous membranes are similarly affected, it is an indication of more or less acute systemic disturbance, and is met with in most fevers. Mucous membranes may also have a bluish tinge, and are then spoken of as *cyanotic* or *dusky* or *livid*. This condition is usually an indication of some interference with the proper oxygenation of the blood, and is due to the reduction of oxyhæmoglobin of the red blood corpuscles to methæmoglobin, often owing to the presence of carbonic acid gas. It is seen in such conditions as extensive bronchitis or pneumonia, and in patent *foramen ovale* in the heart of the newly born.

When the mucous membranes are yellow their condition is called *icteric* or *jaundiced*. This colour may originate from any blood disease which is associated with the breaking down of red blood corpuscles and the liberation of free hæmoglobin, as in cases of azoturia in horses and redwater in cattle. The yellow colour may also be due to affections of the liver in which there is some obstruction to the proper discharge into the intestine of bile, which consequently becomes reabsorbed by the blood with a tendency to yellow staining of all the tissues. The yellow colour in icterus of hepatic origin is usually more vivid than in the former case. The jaundice is most usually observed in mucous membranes which are also pallid, and the colour is most obvious in daylight. In artificial light the yellow colour is not so readily observed. When icterus occurs in an injected mucous membrane the colour is descriptively styled *brick red*. Mucous membranes may also be affected with small hæmorrhagic markings, and these are called *ecchymoses*. When they are small and circumscribed, resembling flea-bites, they are called *petechiæ*. When the markings are long and whiplike they are called *vibices*. The origin of these conditions is somewhat obscure, but they are met with in such affections as purpura hæmorrhagica and some acute fevers, and in pernicious anæmia.

In examining a patient one should observe the condition of all or several visible mucous membranes, and not limit the examination to any individual one. When the abnormal condition observed is present throughout, the condition is obviously a systemic one; while if a single mucous membrane only is affected the condition will be a purely local one and is most likely to be due to some local irritant. The examination of mucous membranes should always be carried out gently, since any violent manipulation may set up a lesion, or at any rate modify the appearance of the existing condition.

The Skin.—In health the skin should be soft and supple, and freely movable upon the underlying structures. The coat should also be glossy and resilient or elastic, and not present a harsh feeling to the touch. A *staring coat*, consisting of a dry harsh condition of the hair, is a common indication of unthriftiness; at other times it is the result of external parasites, such as lice; while in other cases it may be a premonitory symptom of some more serious systemic affection, as in the early stages of fevers. Loss of hair may be an indication either of a local affection limited to the skin as in some forms of mange, or it may be brought about as the result of some chronic debilitating affection. Loss of hair is called *alopecia*. Dryness and moisture of the skin are recognized. In a state of health there is a constant secretion of moisture by the sweat-glands present in the skin, but this evaporates more or less rapidly, so that the skin never gives the impression of being wet. The secretion of moisture may be increased and become visible, either by forced exercise or hot weather or some diseased conditions. Excessive moisture is called *sweating* or *hidrosis*. When very profuse it is called *hyperidrosis*. Increased secretion of the skin as the result of high external temperature or of forced exercise is due to increased circulation through the skin, and is called a *hot sweat*. This phenomenon may be regarded as a normal or physiological attempt to maintain the balance of temperature. *Cold sweats* may occur as the result of acute nervous impressions, the sweat-glands being stimulated through their nerve supply to increased secretion without increased blood supply. Examples of this occur in acute physical pain, and in the human subject also in cases of acute mental stress. It is a debatable point whether the latter can occur in the so-called lower animals, but the writer has observed instances, both in the horse and in the dog, where fright or terror has undoubtedly been associated with droplets of moisture on the skin which could only be regarded as cold sweats. Defective perspiration is called *anidrosis*, and produces the impression of a dry skin. It is somewhat difficult to observe on the general body surface, but may be seen in places which are normally moist, such as the muzzle of the ox and the dog, and the snout of the pig.

Hidebound is the name given to the condition when the skin appears to be closely adherent to the subcutaneous structures, almost giving the impression of being too small for the body it covers. It is due to the arrest in the production of subcutaneous fat, and is therefore one of the results of malnutrition or emaciation from some wasting disease. In this condition it is impossible to raise the skin in folds as may be readily done in a well-nourished animal.

Swellings may be observed in or under the skin. They may be due to either local irritation, or they may be associated with some general systemic disturbance. In the former case the swellings are generally hot and more or less painful. When systemic they are usually soft or doughy when pressed with the finger-tips, and are the result of local dropsy or œdema, as in the case of purpura hæmorrhagica and influenza in horses, and under the chest in traumatic pericarditis in cattle. Local circumscribed swellings (plaques) may be observed in such affections as dourine, urticaria, etc. Local swellings may also occur under the skin due to the presence of gas (emphysema). In the majority of instances this condition is brought about by organisms which may have gained access through some wound.

The *bodily condition* of the patient should also be observed in making the diagnosis. By this is meant the appearance of general nutrition, and a certain degree of rotundity of body, and whether or not the animal is gaining or losing weight. Obviously the best way to determine the latter is to make use of the scales periodically. A distinction should be made between thinness of condition and emaciation. The former may be a leanness quite normal to the individual, as in the case of many cows in heavy milk. Emaciation, on the other hand, is loss of flesh, etc., due to disease or starvation or advanced age. A gradual progressive loss of condition is called *marasmus* or *cachexia*.

Symptoms connected with the Alimentary Tract.—*The Appetite*. Variation in the appetite is one of the first indications of ill-health in a considerable number of animals, in fact it is frequently the first indication to the owner or attendant that an animal is other than perfectly healthy. Defective appetite, however, does not in any way indicate the nature of the disease, and thus may be regarded as a premonitory symptom. Loss of appetite is called *inappetence* or *anorexia*, and the latter term is frequently adopted for convenience as the name of the affection in those cases where an animal is seen before diagnostic symptoms of the pending affection have developed, and in which the owner so frequently insists on being told the name of the affection. The opposite condition of excessive appetite is called *bulimia*. Depraved or perverted appetite, which is sometimes an indication of indigestion, is called *cynorexia*.

Prehension or taking up of food is performed in different ways by various species of animals. Horses, for example, normally collect food with their lips and pass it into the mouth for mastication; in sheep and goats the lips again perform a very prominent part in prehension; consequently affections of the lips in either of these species frequently render it impossible for them to take up food. This inability should not be confused with "anorexia" or lack of desire for food. In cattle the food is drawn into the mouth by means of the tongue, consequently prehension is difficult or impossible in diseases involving the tongue, such as actinomycosis, etc. In swine, dogs, and cats the food is grasped by the incisor teeth and tushes directly.

In partaking of water practically all species except the dog and cat immerse their lips and drink by suction. This is rendered difficult in some paralyses, especially of the seventh cranial nerve, in which case the affected animals have to dip almost the entire lower half of the face. In the dog and cat, liquids are taken in by lapping. In cases of paralysis of the tongue in these animals, or where the free portion of the tongue has been lost, as in the case of dogs with gangrenous glossitis (Stuttgart disease), they learn to drink almost in the same fashion as horses, provided the liquid is given to them in a vessel sufficiently deep for them to immerse the mouth completely.

Mastication. The degree of mastication depends largely upon the nature of the food, and often on the presence or absence of diseased conditions of the mouth and teeth. In the case of dry foods mastication is generally more complete, as might be expected, than in the case of moist or soft foods. It frequently happens, in horses particularly but not exclusively, that, while masticating, the mouth is suddenly opened, and a bolus of food allowed to drop out. This action is called " quidding " and generally indicates some affection of the teeth. Mastication is also painful where there is any acute inflammatory condition of the mouth, and in such cases is frequently associated with dribbling of saliva and frothing at the lips. In fact such painful conditions may actually be responsible for complete indisposition to take any solid food whatever. Grinding of the teeth is often observed, particularly in cattle. It is generally an indication of nausea, and, as a rule, is not associated with any affection of the mouth.

Swallowing is technically known as *deglutition.* Any difficulty of deglutition is called *dysphagia.* This may be met with in inflamed conditions of the throat which frequently result in swelling of mucous membranes, and mechanical interference with the passage of food from the mouth into the gullet. Sometimes, as the result of the obstruction, food material, particularly liquids or semisolids, may be returned through the nostrils, as is sometimes seen in strangles in horses. Dysphagia is also indicated by spasmodic coughing during attempts at swallowing. Difficult deglutition may occur in such affections as tetanus, rabies, actinomycosis, etc.

Vomiting. This is an involuntary expulsion of the contents of the stomach through the mouth or nostrils. It is associated with contraction of the abdominal muscles and the lower cervical (or neck) muscles (retching). Vomiting may take place very easily and freely in dogs, cats, and pigs. It is not uncommon in ruminants, and is extremely rare, though it undoubtedly may occur, in horses. In horses it is not an uncommon occurrence for retching to occur, and to be followed by the expulsion of food material by the mouth or nostrils, but such cases are most frequently due to some œsophageal irregularity such as stricture associated with dilatation.

Foreign bodies in the back of the throat or in the œsophagus frequently produce similar muscular spasms.

The Fæces. The act of expulsion of fæces is called *defæcation.* The character of normal fæces varies considerably in different species, and also frequently in animals of the same species, depending largely upon the character of the food. Frequency of defæcation also varies; it is most frequent in cattle, then horses, averaging from 10 to 15 times per day, whereas in carnivora once or twice daily is a normal frequency. When there is cessation or reduced frequency in the passage of fæces the condition is called *constipation.* Fæces collected in small hard lumps are called *scybala.* In such cases there is usually a thin coating of mucus, which is normal in such animals as the horse and the sheep. In cases of constipation, however, or where the fæces have been withheld for some time, the coating of mucus becomes excessive. *Diarrhœa* is the frequent passage of liquid fæces, and is often met with in irritable conditions of the bowels. When the evacuations are mixed with blood which retains its colour the condition is called *dysentery.* When blood is passed with the fæces, but has become broken down and black, the condition is known as *melœna.* The *colour* of the fæces depends largely upon the character of the food. When there is a deficiency of bile being secreted, the fæces are lighter in colour than normal, and are spoken of as *clay-coloured*; while, on the other hand, when there is an excessive secretion of bile they become bright yellow, and are called *icteric.* Various medicinal agents also tend to modify the colour of fæces, as in the case of iron and bismuth salts, both of which tend to produce blackness of the evacuations.

Tenesmus is straining with reference to defæcation. It is usually an indication of some irritable condition of the lower part of the intestine, and consequently not usually associated with constipation, rather the reverse. It frequently gives rise, however, to a wrong impression on the part of the owner or attendant, who almost invariably considers the straining as a symptom of constipation. One must guard against accepting this subjective symptom without verification by examination.

The Urinary Apparatus. The normal passage of urine is called *micturition.* Any difficulty of urination is called *dysuria.* The passing of excessive quantities is called *polyuria.* Total suppression is called *anuria,* while partial suppression or retention is called *ischuria.* *Strangury* or *stranguria* is that form of dysuria which is painful and spasmodic, the urine being passed in drops or jets. *Incontinence of urine* is the inability to retain urine. It is usually due to some injury or debility of the sphincter of the bladder. The debility in such a case may be of nervous (spinal) origin. Stone in the bladder may also cause incontinence of urine.

An increase in the quantity of urine may occur in some forms of indigestion and in some affections of the kidney, also in the primary stages of fevers, and frequently in convalescence after fevers. Decrease in the amount of urine occurs in hot weather, with sweating, in the acute stages of fevers, and in some chronic kidney affections. The colour of urine very largely depends on the relative proportion of solids and liquids contained. Obviously the larger the proportion of water the paler it will be. In this way the administration of nitrates, being simple diuretics, renders the urine lighter than normal. The normal pigments that are present are probably bile products which have not been excreted through the alimentary tract or which may have been reabsorbed. Where the urine is less copious it becomes dark-coloured owing to the higher proportion of solids compared with water. Certain medicines also tend to alter the colour; for example, carbolic acid, aloes, and rhubarb tend to darken it.

When blood is present in the urine it is called *hœmaturia.* *Hœmoglobinuria* is the presence of free hæmoglobin (colouring-matter of blood) in the urine, as in the case of azoturia in horses. *Albuminuria* implies the presence of albumen from any source in the urine; while *glycosuria* implies the presence of sugar.

In the examination of a patient information may also be obtained by (*a*) palpation; (*b*) percussion; (*c*) auscultation.

Palpation consists in the manipulation of the part or organ with the fingers to ascertain either its consistency, or the presence of enlargement, or the evidence of pain on pressure. Thus one detects the seat of injury by direct examination, as in the case of sprains of ligaments or tendons; also one observes a tenseness of the abdominal muscles in cases of painful conditions of the peritoneal cavity, tenseness of muscles in rheumatism, and rigidity in tetanus, etc. In the small animals, in fact, the whole contents of the abdominal cavity can frequently be examined individually by palpation. In the larger animals, for obvious reasons, the usefulness of the process with reference to the abdominal cavity is more limited, yet one constantly makes use of the method in diagnosing gastric troubles in ruminants with particular reference to the consistency of the contents of the rumen.

Exploration consists in examining a cavity of the body by the insertion of the hand (manual exploration) or of the finger (digital exploration). Thus an examination of the cavity of the mouth, of the rectum, and of the vagina is manual in the case of the larger animals, while it is digital in the case of the smaller animals.

Percussion consists in striking the part to be examined in order to note the sound produced thereby. Percussion may be (*a*) direct or immediate, or (*b*) indirect or mediate. Direct percussion consists in striking the part with the knuckles of the closed fist without any intervening body other than possibly the fingers of the opposite hand. Indirect or mediate percussion is carried out by means of an instrument called a mallet or percussor, and an ivory disc called a pleximeter. The sounds produced may be "resonant" or "dull" according to whether the body or part percussed is hollow or solid.

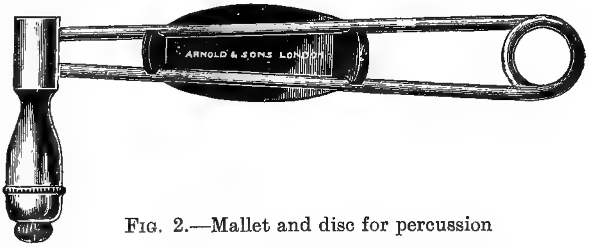

FIG. 2.—Mallet and disc for percussion

In percussing the chest in health, resonance should be clear, particularly over the central portion of the chest wall. It is less resonant as one approaches the shoulder, owing to the presence of more or less thick muscles. The resonance is increased in such conditions as emphysema (broken wind in horses) and in pneumothorax. On the other hand, the sound may become dull or flat where it should be resonant, when the lung becomes consolidated, as in advanced stages of pneumonia. Resonance is also lost with the presence of fluid in the chest, as in a moist pleurisy. In the latter case the dulness is more or less limited to the lower part of the chest.

Percussion of the abdominal wall also assists in the detection of tympany. In cattle in tympanites there is an accumulation of gas in the rumen or first compartment of the stomach which occupies the left side of the abdominal cavity. Percussion here reveals marked resonance.

Percussion is also resorted to in horses to assist in the detection of pus in the facial sinuses.

Auscultation or *sounding* is listening to the sounds produced in or under the part examined, and is resorted to in connection with the chest, the abdominal cavity, and also in the detection of fractures. Auscultation may be carried out either *directly* or *indirectly*. It is direct when the unaided ear is applied to the part being examined. Indirect auscultation is performed with the assistance of certain instruments such as the stethoscope and phonendoscope. These aids to auscultation are very valuable, although to the beginner they appear to complicate the sounds, particularly in veterinary practice, owing to friction sounds caused by the hair on the body. One soon, however, learns to differentiate these sounds from those produced in the deeper structures or cavities. Of the instruments used for this purpose probably the best is the binaural stethoscope with a rubber attachment (phonophore) on the receiver. This latter contrivance minimizes any crackling sound produced by the hair.

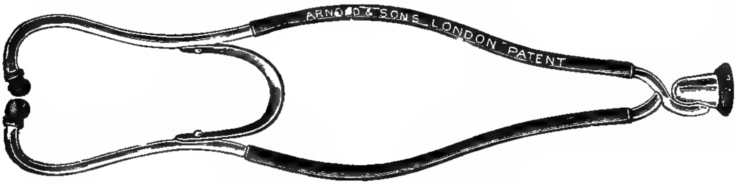

Fig. 3.—Binaural stethoscope.

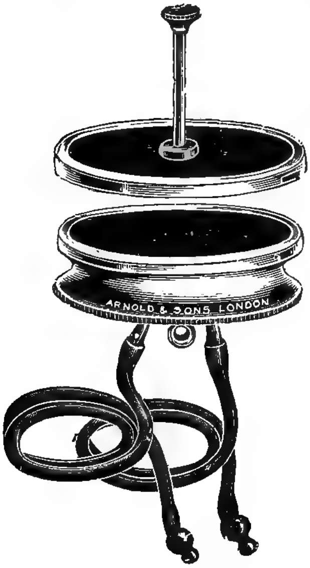

Fig. 4.—Phonendoscope

The phonendoscope may also be used, but it so greatly intensifies the sounds caused by the presence of hair that it is not nearly so serviceable in veterinary practice.

Having examined a patient thoroughly for the discovery of symptoms, one carefully considers them in order to arrive at a diagnosis before treatment can be adopted, and it is obvious that the efficacy of treatment must depend on the correctness of the diagnosis.

Treatment of Disease.—Treatment is divided into preventive and curative treatment. *Preventive treatment* consists of the adoption of various steps with a view to the prevention of the disease. Apart from simple hygiene, preventive measures are only adopted where disease is imminent, and are therefore put into action chiefly to produce immunity or resistance to contagious diseases.

Curative treatment is directed to the restoration of health. It consists in efforts (*a*) to increase the vitality of the patient, and so improve the natural powers of recovery (*vis medicatrix naturæ*); (*b*) to combat the ill-effects produced by the disease, and to stimulate repair of tissue; (*c*) to relieve symptoms which have been set up (palliative treatment); (*d*) to anticipate the course usually run by the disease and thus prevent or minimize the extension of the disease or prevent complications (expectant treatment). Careful nursing is by far the most important part of curative treatment, and its value cannot well be over-estimated.

When treatment of disease is based upon a knowledge of the pathology of the disease, and of the actions of the various agents employed, it is called *rational treatment.* In some cases, however, and unfortunately it applies to many diseases, the true pathology is as yet unknown; but since the effects of the disease are well known, treatment may be and is directed towards the relief of those symptoms by means of nursing and certain medicinal agents whose pharmacological actions are known. Such treatment may still be regarded as rational. On the other hand, when treatment is not based upon a knowledge of the pathology of the disease or the action of the medicinal agents being prescribed, that is known as *empirical treatment* (empiricism or quackery). It will thus be seen that for the practice of rational treatment a correct diagnosis is all-important, and herein lies the danger of amateur treatment of disease.

Termination of Disease.—Disease may result or terminate in resolution or recovery, partial or complete; or it may result in death. Death of the body as a whole is called *somatic* death, as opposed to *necrosis*, which is death of a part still attached to the body. When such a dead part undergoes putrefaction the condition is called *gangrene.*

When death is due to interference with the heart's action it is called *syncope.* This term is frequently limited to sudden death resulting from heart failure, as in the case of shock.

When, however, cessation of the heart's action occurs as the result of exhaustion during a prolonged or debilitating illness the term *asthenia* is used.

Death beginning at the brain is called *coma*. This may be due to circulatory disturbances in the brain, such as venous congestion, or as the result of various narcotic poisons. Coma is indicated by stupor or partial or complete loss of consciousness, and is sometimes preceded by a stage of excitement. It may also be caused by direct injury to the brain, or by pressure of such things as tumours or abscesses or blood-clots from hæmorrhage. Death in coma is due to the interference with vital functions which are under control of the brain centres involved in the diseased process.

Death commencing with the lungs is called *asphyxia* (apnœa), and is usually convulsive. It is practically the same thing as suffocation, and may therefore occur as the result of any condition which interferes with the function of respiration, such as extensive pneumonia, obstruction to the respiratory tract, drowning, strangulation, or paralysis or rupture of the diaphragm. The convulsions of asphyxia are alternately inspiratory and expiratory. As the blood cannot pass through the lungs the right side of the heart becomes over-distended, and also the superficial veins which can be very readily observed. Another result of this is the production of a jugular pulse, while the visible mucous membranes become bluish (cyanotic). After death from asphyxia the right side of the heart is found to be over-distended with blood, the lungs congested, and the bronchial tubes and trachea full of froth.

Immunity.—When an animal cannot be affected by the germ of a particular disease it is said to have immunity against that particular disease. Immunity to contagious disease may be *natural* or *acquired. Natural immunity* is the inherited resistance against infection by the causal organisms of a disease possessed by an individual animal or by a species of animals. It not infrequently happens that an animal is exposed to infection along with other animals and escapes while the others become affected. The natural immunity of a species is, however, more notable, as, for example, that of cattle against glanders—a serious fatal disease of horses—and that of horses against the very serious bovine disease, cattle-plague. No satisfactory explanation of this phenomenon can be offered.

Acquired immunity is generally due to the animal having survived a natural attack of the disease in question, or to its having been treated in some way or other, as by vaccination, against that disease. The resistance in such cases is ascribed to "antibodies" circulating in the blood and which have been produced by the reaction of the tissues of the body. The antibodies afford protection only against the particular organism responsible for their production; this immunity is therefore specific.

Acquired immunity may be "active" or "passive." *Active immunity* is the form which results either from the recovery from a natural attack of the disease, or from vaccination with living (though possibly attenuated) bacteria of the disease. *Passive immunity* is that form which results from the injection of the products of a bacterium, or from the injection of germ-free blood serum from an animal that has survived a natural attack or has been treated with the bacterial toxins. The immunity of an animal can be raised by a succession of injections of virus or toxin of increasing strength, so that the individual can withstand the inoculation of huge doses of infective and highly virulent material. Such an animal is said to be hypervaccinated or hyperimmunized, and its blood yields the so-called hyperimmune serum, which is highly charged with antibodies and is usually employed in the production of passive immunity, Active immunity is much stronger and more lasting than passive immunity, but an interval is required after the introduction of a vaccine for the development of protection. On the other hand, the passive immunity conferred by the injection of a so-called "antitoxic serum" is more transient, seldom lasting more than a few weeks, but it is *immediately* acquired after the introduction of the serum.

It is important, therefore, to distinguish between a vaccine and an antitoxic serum. A *vaccine* actually contains the organisms of the disease living (though possibly attenuated) or dead, and prepared either directly from the diseased tissues containing them or from artificial cultures. If the vaccine is prepared from materials obtained direct from the patient to be treated it is called an "autogenous vaccine." Vaccines consisting only of dead organisms have been called *bacterins* and *phylacogens* to distinguish them from those containing living organisms. An *antitoxic serum* contains no organisms and so cannot possibly set up the disease. It is, however, highly charged with antibodies, is capable of producing immediate passive immunity, and in some cases may actually exert a curative effect in an animal suffering from the disease if injected in considerably larger doses.

Immunity is sometimes produced by the combined use of an antitoxic serum and a virulent or potent vaccine, the former providing some protection against the latter, thus reducing the danger of setting up the disease while producing a strong degree of active immunity. This procedure is called the "simultaneous method" of immunization.

G. H. W.

CONTAGIOUS DISEASES

HISTORICAL

The existence of diseases of a contagious character appears to have been recognized from the dawn of history. Thus the ancient Persians and Israelites describe leprosy in man; Isocrates, consumption. The presence of plagues among cattle in ancient Egypt several centuries before the Christian era can be made out from the rare papyri recently discovered. Among the ancient Greeks there is but little doubt that important observations were made on the diseases of the domesticated animals, but, unfortunately, few of their writings survive. Aristotle (333 B.C.) deals with a number of diseases affecting farm animals, and these include in all probability anthrax, glanders, contagious bovine pleuro-pneumonia, foot-and-mouth disease, and rabies. Further, this writer argues that the healthy were liable to contract disease through proximity with the diseased.

Roman writers, living during the century before Christ, and deriving undoubtedly the greater part of their information from the earlier Greeks, also deal with these diseases and advance remarkably sound views from the modern standpoint with regard to the nature of the contagion. Thus, Varro ascribed the transmission of disease to the presence of minute living particles which were ingested or inhaled. Again, Columella gives clear accounts of plagues in cattle, mange in horses, sheep-scab, sheep-pox, and rabies, and their contagious nature is well understood; for the first time in history the segregation of the sick is insisted upon: "The diseased must be separated from the sound, that not so much as one may come among them which may with the contagion affect the rest."

The distinguished veterinary writers of the early Byzantine period—notably Apsyrtus, "the father of veterinary medicine" (fourth century A.D.), Chiron, and others, whose writings were collected during the early tenth century by the Emperor Constantine the Seventh, and destined to leave a permanent imprint, especially after the Renaissance, on veterinary literature right up to modern times—dwelt at great length on the clinical manifestations of certain contagious diseases. Accounts of outbreaks of disease, described as "malleus" among horses, leave no doubt that glanders and farcy, and probably strangles, and some other affections included under that term were of extremely common occurrence in those days. Chiron notes with regard to malleus: "It is a pestiferous disease of the body . . . wicked and incurable. . . . If it gets hold of a herd . . it consumes them all." A detailed account of the disease now known as dourine and its transmission by copulation is also given by the latter author.

A short time before the fall of the Western Roman Empire, Vegetius (A.D. 450–500), a great collector of information on animal diseases, also described the malleus group of disorders, and included accounts of plague or pestilence in cattle, embodying most probably rinderpest and foot-and-mouth disease. The measures in force in the Roman states for suppressing outbreaks of contagious diseases are also outlined.

During the course of several centuries subsequently no advance was made in the study of disease. The Arabs were reputed to be well versed in knowledge of animal diseases, but their information was of Greek or Byzantine origin, and they added nothing. Otherwise all through the Dark Ages the most intense ignorance prevailed with regard to their nature. Fostered to a great extent by the pernicious influence of the church in crushing all attempts at obtaining knowledge, the human mind confined itself to the treatment of all diseases by filthy remedies and incantations even for animals, with no regard to studying their origin or clinical course. This tendency to enshroud the subject of disease with a thick cloak of superstition has, moreover, been always characteristic of human beings in all ages, in all countries, and of all classes. In the days of Vegetius "divine displeasure was invoked to explain the ruthless destruction of animal life, and bankruptcy of owners"; he pleads against this doctrine as one which can "only be held by fools" (Smith). The same attitude can be seen displayed even to this day by uncivilized African tribes and Turks, who view with the greatest complacency the destruction of their herds by rinderpest and impending famine. "Allah gives, Allah takes . . ." Human impatience to obtain a "cure" for all affections is also in quite recent evidence, as may be gathered from the following remarks quoted from Montgomery in alluding to the discovery of the cause of the formidable Indian disease known as surra by Evans in 1880: "One almost reads a note of censure in the remarks made by the

Lieutenant-Governor of the Punjab on Evans' Report, 'that whilst recognizing the importance of the discovery, he regretted that no cure had been found for the disorder.'"[1]

Thus, with rare exceptions, few original observations were made on animal diseases during the Middle Ages. There is every reason to believe, however, that plagues affecting farm stock were by no means uncommon. For example, the French writer on sheep farming, Jehan de Brie, in the fourteenth century describes the occurrence of scab and liver rot in sheep in France, and for the first time mentions flukes as the cause of the latter disorder; in 1552 the same affection was the cause of severe outbreaks among sheep in Holland. Towards the end of the thirteenth century, wool manufacture, which had become the staple industry of England, was nearly threatened with destruction owing to the ravages caused by sheep-pox, a disease introduced into Northumberland with some sheep imported from France. The last-named disease is also mentioned by an English writer, J. Fitzherbert, in a work published in 1523, and the necessity for isolation is stated; further, in dealing at length with so-called "rot" in sheep, he says that all kinds of flooded land are bad for sheep, and "white snails be ill for sheep in pastures and in fallows"—a remarkable statement made 360 years before the white snail was definitely convicted of acting as intermediate host in the transmission of the parasite of the disease. "Lengsought" of cattle, probably contagious bovine pleuro-pneumonia, is described by the same author; the affected were isolated, as the disease was recognized to be contagious.

After the Renaissance, partly owing to the dissemination of the writings of the early Byzantine writers, and also partly owing to increased travelling and interchange of animals for breeding purposes, with consequent importation of contagious disease, the study of animal diseases made slow but gradual progress. Thus, among other diseases, we find rinderpest appearing in Western Europe, and in 1711 State veterinary regulations were formulated in Prussia, and in 1714 in France, for the control of the disease. Later, in 1744, we hear of Dodson in England, and Camper in 1770, performing experiments with regard to transmission of and protective inoculation against the disease; for the latter purpose nasal secretions were injected under the skin of the tail or other places.

With the establishment of veterinary schools in Western Europe, commencing with the institution of the Lyons school by Bourgelat in 1763, the study of animal disease became the pursuit of highly-trained observers. At the beginning of the nineteenth century the grosser parasites causing widespread losses among stock were clearly described by Rudolphi, Zeder, and others, and a generation later by such distinguished writers as Gerlach, Leuckart, and Delafond. Again at the commencement of the second half of the century we find the experimental method of research, destined later to give such remarkable results in furthering our knowledge of animal diseases, employed largely and developed by members of the brilliant French school, such as Bouley, Trasbot, and Chauveau, and in Germany by Bollinger and others.

Our present knowledge with regard to contagious disease may, however, be said to rest mainly on the fundamental researches of Pasteur, whose observations, commencing about 1860, on the part played by minute living organisms in bringing about decomposition of organic material, later in the production of well-known diseases, and again the methods for the protection of the animal body against fatal infection by these germs, were not only in themselves of great economic value but also furnished the guiding principles on which future investigators were to work in an endeavour to determine the causes and methods of control of all other diseases of a contagious or infectious character. At about the same time Robert Koch, largely by the employment of methods for studying minute organisms in pure culture, also published observations which incriminated specific germs beyond doubt as the cause of certain diseases; and further he and his pupils discovered the causal organisms of a number of other common and serious diseases, both human and animal.

The views held prior to the latter half of the preceding century and even at the present time by the untrained mind with reference to the origin of disease need only be briefly discussed here. Single cases of disease could generally be assigned to some definite cause, but in order to explain the occurrence of a certain disease simultaneously among a large number of men or animals it became necessary to invoke various hypotheses in order to explain the conditions. First, there was the so-called "humoral" theory, in which certain changes were said to have been brought about, the "humours" or body-fluids resulting from faulty nutrition, exposure, overstocking, atmospheric conditions, or other ill-defined causes. The farcy lesions of glanders, mange in horses, and scab in sheep were held until comparatively recently to be set up by these means. Secondly, there was the so-called "miasmatic" theory in which it was stated that certain diseases were conveyed by some "miasma" or mysterious disease-bearing or pestiferous substance in the air. Both these theories are apparently embodied, for example, in the explanation given by Chiron (about A.D. 400) of the malleus group of disorders. As quoted by Smith, Chiron states that "it is produced by cold, fatigue, plethora, bad hay, and other causes have been assigned. But he insists that the actual exciting cause is the pestiferous, hot southerly wind from Africa. It is this which conveys the infection, which is taken in by the lungs and 'born in the blood.'" The importance of guarding against these "miasmas," which were held to be composed largely of emanations from the bodies of dead animals, may be gathered from the following sixteenth-century rhyme (quoted from Smith):

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[1] *Journal of Tropical Veterinary Science*, 1908, vol. iii. p. 301.

> Whatever thing dieth, go burie or burne,
> For tainting of ground or a worser it turne,
> Such pestilent smell of a carrenly thing,
> To cattle and people great peril may bring.

Further, the common name given to the widespread human disease malaria (*mal aria*, bad air) owes its origin to this theory.

As pointed out before, some diseases have long been recognized to be, at least to a large extent, contagious, that is, conveyed from diseased to healthy animals. Thus Albert of Cologne (1206–1280), who appears to be the first writer on veterinary subjects to consider systematically this question, deals with "'corrupt' blood, and then describes three modes by which the contagion might be conveyed to other animals. He classifies these as due: (1) to inoculation by bites or injuries; (2) actual contact with diseased animals, or the occupation of places previously used by them; (3) the respired air from the sick, which he regarded as the most important of the three" (Smith). An English writer of the same period (Bartholomew Glanville) gives the following account of the transmission of rabies: "The biting of the wood hound is deadly and venomous, and such venom is perilous, for it is long hidden and unknown, and increaseth and multiplieth itself, and is sometimes unknown to the year end, and then at the same day and hour it cometh to the head and breedeth frenzy" (Smith). Fracastor (1546) again divides contagion into three kinds, viz. *contagio per contactum*, *contagio per fomites* (*i.e.* by clothing, bedding, etc.), and *contagio per distans*; contagion emanated only from a sick man or animal, miasma from dead material; some diseases were distinctly contagious, others eminently miasmatic.

Matters remained in this state until after the middle of the nineteenth century. Henle, writing in 1853, states that with each fresh epidemic in Europe the medical world became divided into two camps, viz. those who upheld the miasmatic theory and those supporting the contagion theory.

From the earliest times it had been recognized that the smallest trace of infective material was capable of setting up the disease, and thus contagion came to be reckoned to be one of three things, viz. poison, ferment, or living object. The difference between the action of chemical poisons and disease-producing substances was recognized by Fracastor in 1550. The opinion that contagious disease was set up by minute living organisms seems to have been first expressed by Varro, who says, "it is also to be noticed, if there be any marshy places, that certain minute animals breed there which are invisible to the eye, and yet, getting into the system through mouth and nostrils, cause serious disorders."

With the invention of the microscope towards the beginning of the seventeenth century it became possible to study forms of life too small to be identified by the unaided eye. Bacteria were first recognized with certainty by van Leeuwenhoeck towards the end of the century in saliva, diarrhœic fæces, and other secretions and excretions. They were later described by other observers in the excretions of patients suffering from contagious disease.

In 1835 a disease of silkworms known as "muscardine," up to that date reckoned to be of a most distinct contagious-miasmatic character, was investigated by Bassi, who found the cause to be a mould affecting the young adult insect and the affection to be spread by the spores of the mould found on the surface of the dried-up dead insect.

After carefully weighing the existing evidence on the subject, Henle, in 1840, came to the conclusion that the cause of contagious or infectious diseases must be looked for in minute living organisms or fungi; he thus propounded what may be called the Germ Theory of Disease, and further formulated certain strict conditions which a suspected micro-organism must fulfil before it can be held to be the cause of the disease, viz. (1) the organism must be constantly associated with the disease; (2) it must be isolated and studied; and (3) when introduced into animals susceptible to the natural disease it must reproduce the disease in its original form. For a number of years these conditions were lost sight of by investigators, until they were again taken up seriously by Robert Koch (1876) in his studies on anthrax.

In the meantime, however, minute parasites were discovered in connection with a number of diseases. Gruby (1842) demonstrated a form of mould (trichophyton) in the human affection known as "barber's itch," and the following year Malmsten discovered the same class of organism in ordinary ringworm of man. Later

Bazin (1853) described this kind of fungus in ringworm of the horse, and Gerlach (1857), in an important work, showed the parasitic nature of ringworm in cattle. The last-named author in the same year studied and described the minute acarian parasite which is the cause of mange in horses, and in the following year Delafond discovered the same kind of small mite to be the cause of scab in sheep.

In 1850 Davaine and Rayer, in 1855 Pollender, and shortly afterwards Brauell, demonstrated the presence of rod-like bodies (bacilli) of a special kind in the carcases of animals dead of anthrax; Brauell found these rods also in the blood of living animals suffering from the disease. Afterwards, Delafond (1860) described the plant-like character of these small rods, and then Davaine (1865) showed that inoculation of blood containing these rods, even when very highly diluted, set up the disease in healthy animals, whereas inoculation of blood not containing such rods produced no result.

The study of minute living organisms now became a matter of the highest importance, and principally as the result of Pasteur's researches on the decomposition of organic matters; this subject had been investigated very incompletely by a number of previous authors. Pasteur showed that decomposition or putrefaction was constantly associated with the presence of living bodies or bacteria. Further, if a distinctly putrescible liquid, such as blood or blood serum, was withdrawn from the body so as to exclude admission of outside contamination, and kept in sealed flasks which had been previously heated so as to kill all bacteria inside the flask, then this liquid remained free of germs indefinitely, and decomposition did not set in. The same result could be obtained if a liquid such as broth were placed in the flask and heated sufficiently high (110° C.) to kill all germs within it. Directly, however, a trace of air was admitted into the flask decomposition set in with abundant multiplication of germs. Putrefaction was thus set up by the admission of something present in the air entering the flask; Pasteur then proved that bacteria are always present in great numbers in the atmosphere as well as in soil and water, and that it was the admission of these minute organisms from the outside world which set up putrefaction. Again, it was shown that the souring of sugar-containing substances such as beer, wine, and milk, the rancidity of butter, and the putrefaction of flesh were all brought about by the action of certain kinds of small plant-like bodies; in the case of the organism producing rancidity in butter it was shown that it would only grow in an atmosphere deprived of oxygen. The most important fact was that these organisms had been introduced from an outside source, and had thus multiplied from pre-existing organisms. Moreover, their presence was an indispensable condition to the production of the changes. The theory of their spontaneous origin was thus disposed of and also the hypothesis advanced, especially by Liebig, that putrefaction was a disintegration of nitrogenous compounds brought about by chemical or chemico-physical agents acting in the manner of ferments.

Klebs (1872) and others showed that bacteria were always present in the pus of infected wounds and also were instrumental in the production of the changes seen in so-called blood-poisoning (pyæmia, septicæmia). These facts, together with the researches of Pasteur, induced Lister to adopt his method of wound-dressing which consisted in the destruction of the minute living organisms by means of chemical agents known as antiseptics, a method destined to revolutionize the art of surgery.

The solution of the problem as to the existence of specific kinds of bacteria was furnished by the fundamental work of Robert Koch in his work on anthrax published in 1876. Ferdinand Cohn (1870) had already shown that the anthrax organism was capable of developing into very resistant forms known as spores, when conditions became adverse for its further growth. These spores, which were difficult to recognize under the microscope, might remain dormant for a long time and sprout out again into the growing or vegetative form directly conditions of temperature and nutrition became favourable. Koch followed under the microscope the development of the bacillus from spore to spore and showed that it was only by the inoculation of the bacillus or its spore, and not by other bacteria, that anthrax could be set up in animals. In cases where it was thought that the disease had been set up by means of a fluid free of spores, it was shown that one had, however, unconsciously injected the bacilli or spores. The so-called "miasmatic" affection of cattle or sheep on pastures could thus be explained by the distribution of spores on the ground. Outbreaks could be prevented by arresting the development of spores from the carcases of dead animals. This was possible if they were buried deep enough so that the bacilli did not find a temperature of over 15° C. which is necessary for spore formation. A couple of years later Koch proved the specificity of the micro-organisms in the case of a number of other pyæmic and septicæmic animal diseases, such as mouse septicæmia and rabbit septicæmia.

A few years later Pasteur and his pupils by their studies on the biology of the organisms causing such diseases as fowl cholera (1880), anthrax (1881), and swine erysipelas (1882), and on the virus of rabies (1881–1889) discovered means by which their virulence or disease-producing capacity could be so weakened or modified that the symptoms occurring after their injection into animals were very slight, but set up as a consequence immunity towards subsequent natural infection. It was thus that the groundwork of immunity towards the contagious diseases became established.

With the perfection of methods employed for the study of bacteria, the causes of a still greater number of the contagious or infectious diseases were discovered. The improvement of the microscope mainly consisting in the use of accurately computed achromatic lenses capable of high resolving powers, the introduction of homogeneous immersion lenses and of the Abbé condenser enabled workers to detect and study details in the structure of the more minute disease-producing germs. Again, the introduction of solid culture media into routine bacteriological study by Koch provided means of obtaining surface growths from single bacteria implanted on to them; from the single visible masses of growth or colonies obtained in this way one could carry on a strain of the original single germ in a state of purity. Koch first employed potato and then gelatine with the addition of various substances such as aqueous humour and serum, and later on serum coagulated by heat; the meat-broth peptone gelatine now generally employed was introduced by Löffler, and agar by Hesse. Methods of staining bacteria also greatly increased their visibility under the microscope and enabled one to appreciate their structure more closely. The so-called basic aniline dyes, first introduced for the purpose by Salomonsen (1876) and tried by Koch (1876) and Ehrlich (1877), proved to be of inestimable value. Drying and fixing of bacteria before staining were first adopted by Koch and Ehrlich. A specific staining reaction for the tubercle bacillus was discovered by Koch (1882); Gram (1884) introduced a method of staining which shows up certain bacteria only; the cilia of bacteria were first stained by Löffler (1890).

Among the more important discoveries of diseases in animals having specific bacteria as their causal agents may be mentioned, in addition to those referred to above, the glanders bacillus by Löffler and Schütz in 1882; the tubercle bacillus, which was first observed by Bollinger (1875), and Feser (1876), and studied by Arloing, Cornevin, and Thomas (1879–1884); the bacillus causing abortion in cattle by Bang in 1896.

Simultaneously with the development of our knowledge regarding the pathogenic bacteria—unicellular organisms which on account of their biological characters are usually classed with the lowest forms of vegetable life—the disease-producing capacities of the lowest forms of the animal kingdom, viz. the protozoa, were also revealed. The parasite which causes outbreaks of diarrhœa and wasting among young rabbits was observed as far back as 1839 by Hake, again in 1845 by Remak, and studied in 1879 by Leuckart. Protozoa of a similar nature have been found to be the cause of outbreaks of intestinal troubles in some places, especially in birds and in cattle. By far the most important kinds of protozoa are, however, the trypanosomes and the piroplasms. Trypanosomes were found as harmless parasites in the blood of the rat by Lewis in India in 1879. The following year Griffith Evans described the occurrence of a similar organism in the blood of horses, mules, and donkeys suffering from surra in the Punjab. A number of years later (1894) Bruce in Zululand discovered a similar organism in the blood of oxen suffering from nagana or tsetse-fly disease. Various species of trypanosomes were later described as the causes of a number of fly-transmitted diseases in tropical countries, and of sleeping-sickness in man. The piroplasm which is the cause of red-water or Texas fever in cattle was first seen by Babes in 1889; and a year later Smith and Kilborne in America published their classical description of the parasite and of its transmission by the tick. Biliary fever in horses, malignant jaundice in dogs, and East Coast fever in cattle were then proved to be caused by similar parasites, part of whose life-cycle was passed in the bodies of ticks. A somewhat similar blood parasite was shown to be associated with human malaria in 1881 by Laveran.

There remained a number of very important animal diseases, such as foot-and-mouth disease, rabies, rinderpest, and contagious bovine pleuro-pneumonia, in which no organisms could be demonstrated as the cause of the affections, although there was every reason to suppose from experimental and clinical studies that they were caused by such. It had already been shown that in the case of a disease of the tobacco plant the virus was so small as to be able to pass through the pores of an earthenware filter. In 1879 Löffler and Frosch showed this to be the case with the virus of foot-and-mouth disease. The organism was thus so minute as to elude detection with the highest powers of the ordinary microscope. In certain conditions it was later proved that the viruses of pleuro-pneumonia of cattle, infectious anæmia of horses, rinderpest, the variolæ, swine fever, rabies, fowl plague, South African horse-sickness, and some other less important diseases could pass through a filter of diatomaceous earth (Berkfeld filter), or even in some cases through the less porous porcelain filter (Chamberland filter). Special methods of cultivation have, however, enabled one to obtain growths of the viruses of contagious bovine pleuro-pneumonia and of rabies; the cultivated forms assumed dimensions which were quite visible under high powers of the microscope. The above so-called ultra-visible viruses are thus better known as filterable viruses, inasmuch as the employment of improved methods may yet place them within the range of visibility. Moreover, the invention of the ultra-microscope has given rise to high hopes in studies of this kind.

From the above brief historical survey it will be seen that the study of disease—and especially of contagious or infectious disease—is intimately wrapped up with the biology of the lower organisms. Thus, although the main facts already disclosed regarding disease are relatively simple, yet these pathogenic organisms present such a complexity of detail in character among themselves that the investigation of disease with a view to its eradication or control necessitates beforehand a prolonged and intensive training in the nature of the minute organisms as well as in the effects of disease upon the host.

J. T. E.

STRANGLES

Febra Pyogenica

Strangles is an acute specific contagious disease of horses, asses, and mules, characterized by catarrh of the mucous membranes of the pharynx and the upper air passages, together with suppuration of the neighbouring lymphatic glands and occasionally those of remote parts of the body.

Etiology.—The lesions of strangles are due to the streptococcus of strangles (Schütz), which can always be found in the catarrhal discharges, and also in practically pure culture in the form of chains in the pus from the affected lymphatic glands. The organism is morphologically and culturally indistinguishable from other streptococci which may be found in suppurative lesions in conditions other than strangles. Although this streptococcus is invariably present in the case of strangles there is some doubt as to whether it is actually responsible for the contagious nature of this disease. It is quite probable in the view of many authorities that the real cause of contagion is an undiscovered virus, possibly an ultra-visible organism, and that the lesions of strangles are produced by the streptococci in the presence of that virus.

The predisposing causes include: *age*, in that it most frequently affects young horses, which contract the affection usually in a more serious form than adults—this may be due to the fact that many adults have already suffered when young from the disease and developed a certain degree of resistance; the *congregating of young horses together* as in dealers' stables, remount depôts, etc.; *ill-ventilated stables*; and *over-exertion of young green horses.* Susceptibility appears to be increased by damp cold weather, and there is said to be a *seasonal influence,* the affection being most frequently met with in the first three months of the year. These predisposing causes, however, are not of first importance, the all-important one being exposure to infection, since there is no such thing as a case of sporadic strangles, and every outbreak must be attributable to a previous case of the disease.

The chief medium of spread is therefore an animal affected with strangles or recently recovered from it. It has been stated that in rare cases animals that have apparently recovered from strangles and show no symptoms of the affection may still remain carriers for at least several months.

Infection may take place by inhalation, by ingestion, and possibly by inoculation, as in the case of erupted teeth and operation wounds. It has also been stated to have been conveyed during coition from an affected stallion to mares. Infective material may be conveyed by soiled forage, litter, mangers, watering-troughs, buckets, nose-bags, sponges, bridles, head-stalls, etc., while infection may also be contracted during transport in railway horse-boxes, on board ship, in stables and market towns where numbers of horses may be put up on market day, and even in the shoeing forge.

The *period of incubation* varies from three to ten days, while the average *course* of the disease in a typical case is from four to six weeks.

The *mortality* is very low, and averages from 1 to 2 per cent, although in some virulent outbreaks a higher death-rate may be met with. Although the mortality from this affection is not very high it is one of the most serious diseases of horses to be met with, particularly owing to the loss of condition of the horses, the length of time thrown off work, and the serious nature of some of the sequels such as roaring and whistling, etc.

Lesions.—The almost constant lesions consist of catarrh of the nasal chambers, the pharynx and larynx, and sometimes also the bronchi. From the affected mucous membranes a thick creamy pus is produced. The streptococcus, after penetrating the mucous membrane, usually passes by way of the lymphatic vessels to the adjacent lymphatic glands. In the majority of cases the submaxillary lymphatic glands under the jaw are affected, while in a considerable number of cases the pharyngeal lymphatic glands at the side of the throat are involved, and more rarely the subparotideal lymphatic gland under the base of the ear. In these glands abscesses are produced which almost invariably burst spontaneously, liberating a thick creamy pus, blood-streaked. The gland then slowly returns to its normal condition. Occasionally the abscesses of the pharyngeal glands burst inwardly and the pus may pass into the larynx and trachea and set up bronchitis, or it may be swallowed and set up secondary lesions in the abdominal organs. The guttural pouches may be affected by an extension of catarrh up the Eustachian tube from the pharynx. The commonest abdominal lesion consists of abscess formation in the mesenteric lymphatic glands. Less common seats of lesions are the prepectoral and bronchial lymphatic glands, the lungs, the pleura, the pericardium, liver, spleen, kidneys, and the skin, while more rarely abscesses may be met with in intermuscular situations, joints, tendon sheaths, bones, and brain. In each situation the streptococcus can be readily demonstrated. The rarer lesions are usually secondary and due to the organism being conveyed by the blood stream.

Symptoms.—Strangles may run either a "typical" or an "atypical" course.

In an ordinary typical case the first signs are a slight dulness and a rise of temperature to 103°-4° or 5° F., which fluctuates during the course between 102° and 105° F. The horse is very depressed, hangs his head down and only picks at food. The pulse becomes soft and sluggish, but is normal in frequency. The nasal mucous membranes are red and injected or sometimes mottled in appearance, and there is at first slight watery nasal discharge, which soon becomes thick and yellowish-white or even greenish in colour, very abundant and bilateral. The conjunctiva is injected and catarrhal, causing a varying degree of discharge from the eyes. There is also a slightly sore throat and an occasional cough.

A swelling of the submaxillary glands then begins with a little heat and tenderness, and in the course of a day or two it becomes larger and firmer, hot and painful, and the intermaxillary space may become completely filled. The swelling gradually becomes softer at one spot towards the centre, indicating the pointing of the abscess, and if not lanced it soon bursts spontaneously, discharging a thick creamy-white pus—at first blood-streaked—containing pure cultures of the streptococci in chains. On the bursting of the abscess the temperature rapidly falls, the patient becomes brighter and begins to feed better, and from this point recovery generally commences. The abscess cavities heal up by granulation arising from the walls of the abscess.

When the pharyngeal glands are affected the swelling in the region of the throat may cause difficulty in swallowing and set up audible respiration or roaring; water and liquid food come back through the nostrils and no solid food can be taken. The roaring or dyspnœa may become very intense and may even set up asphyxia. After the bursting of the abscesses in this situation the relief is very pronounced. During the course of the affection the extremities become œdematous and puffy, and the bodily condition is very considerably reduced.

Various *complications* may occur during the course of strangles. There may be a necrosis of the nasal mucous membrane giving rise to ulceration and to epistaxis or bleeding from the nose. In other cases the catarrh of the pharynx may extend up the Eustachian tube to the guttural pouches, setting up empyema with swellings under the base of the ears and the sides of the throat. This may result in a chronic nasal discharge—one form of nasal gleet. When recovery occurs in such cases the pus retained in the guttural pouches becomes inspissated and formed into small brown semi-translucent concretions called *chondroids*.

The catarrh of the pharynx and the larynx may extend down the trachea to the bronchi, setting up bronchitis or broncho-pneumonia, while at other times when the abscesses burst inwardly considerable quantities of pus may pass down the trachea and set up gangrenous pneumonia, which is invariably fatal. When the pus is swallowed the mesenteric lymphatic glands are likely to become the seat of abscesses, but these glands are more likely to be affected in atypical cases rather than as complications of a typical case.

If metastatic lesions occur as the result of the blood stream being invaded with the organisms, abscesses may be set up in the lungs, liver, spleen, kidneys, and the brain, and when this is the case the patient rapidly becomes weaker, the temperature acutely raised and death follows in a few days. Where the brain is involved death is preceded by meningitis and encephalitis with irregular gait, intense depression, and often blindness.

Joints and tendon sheaths may also become the seats of abscesses; one or more limbs may be involved and the affected areas become swollen and extremely painful. In the majority of cases the abscesses burst, while at other times the lesions recede and leave a permanent thickening of the tendon sheaths with contraction, thus raising the heel from the ground.

If the mammary gland in the case of a mare becomes affected the disease may be transmitted to the foal. One or both halves of the udder may become involved, causing a more or less acute and painful form of mammitis; the supra-mammary lymphatic glands are swollen and painful, and may also be the seats of abscess formation.

Secondary cutaneous lesions are sometimes met with, and these may take the form of small pustules resembling acne diffusely distributed over the head and neck and chest-wall. At other times the extension from the primary lesion may take place along the lymph vessels, causing cording of the lymphatics and the development of small abscesses somewhat resembling glanders farcy and epizoötic lymphangitis. Microscopic examination of the pus, however, readily distinguishes strangles from those conditions which also usually run a chronic course.

Atypical forms of strangles are not infrequent. The commonest is met with in adult horses and consists of nasal catarrh with slight swelling of the lymphatic glands but without abscess formation. In the nasal discharge the streptococci are easily demonstrated. In spite of that fact, however, there is some doubt as to whether any catarrhal affection should really be called strangles in the absence of abscess formation in the glands, but as the streptococcus in these cases is indistinguishable from the streptococcus of strangles it is a much safer proceeding to regard the condition as being strangles in order that the necessary precautions should be taken against contagion.

The base of the neck in front of the shoulder is not an infrequent seat of strangles abscess involving the inferior cervical or prepectoral lymphatic glands. These are sometimes involved without any symptoms of catarrh of the upper air passages having been observed. At other times it is secondary to them. A very large swelling results which may attain to the size of a football, and the obstruction of the lymphatics and the pressure on the jugular veins may bring about œdema of the head and neck. Œdema of the fore-limbs and under the chest also follows. In this condition the appetite is capricious and the temperature is raised to 105°-106° F. The swelling is tense and painful for several days, gradually increasing in size and at last pointing or fluctuating in one spot. The contained pus is under tense pressure and when the abscess is lanced a copious jet of creamy pus may be thrown out several yards. The amount of pus frequently exceeds a gallon. When the abscess is evacuated the symptoms rapidly subside and the patient usually makes a complete recovery under appropriate treatment.

Another seat of irregular or atypical strangles is the pleural cavity, resulting in the production of a suppurative pleurisy and empyema of the chest. One or both sides of the chest may be affected, the temperature and general symptoms being somewhat fluctuating. This purulent form of pleurisy is not so painful as the common form of sporadic pleurisy. When the condition is bilateral, respiration becomes extremely difficult and the patient may die of asphyxia. When it is limited to one side of the chest, recovery, though rare, is possible with adhesions of the lung to the chest wall.

At other times a pneumonia or pericarditis may be met with, due to the same streptococcus, but giving rise in each case to symptoms which are not in any way diagnostic of strangles. It is only on post-mortem examination that the identity of the affection is established in these cases.

Abdominal Strangles.—A common seat of irregular strangles in horses is the mesenteric lymphatic glands. As already stated, these glands may become involved secondarily to primary strangles affecting the throat, but it may also be met with as a primary condition. It gives rise to a subacute affection with fluctuating temperature, general depression, and diarrhœa with great emaciation—occasionally there is an absence of fæces. The appetite is capricious and the temperature fluctuating. In some cases the enlarged glands may be felt in the roof of the abdomen on examination per rectum. The condition may prove fatal in ten to fourteen days, or it may assume a chronic form which extends over many weeks. In the latter cases apparent recovery often occurs, to be followed by relapse with progressive emaciation and occasional attacks of colic.

The majority of cases of abdominal strangles are fatal owing to bursting of the abscess into the peritoneum setting up a purulent peritonitis. On rare occasions an adhesion occurs between the abscess and the bowel wall, the abscess bursting into the intestine and terminating in recovery. At other times adhesions are formed between it and the abdominal wall and it may burst outwardly through the flank.

Sequels.—Probably the commonest sequel to strangles is the development of *roaring* or *whistling*, which is said to occur in not less than 20 per cent of animals that have recovered from the disease. This may be the result either of chronic catarrh of the mucous membranes or it may be due to the effects of the toxins on nerve-endings or even on nerve-centres. On the other hand, strangles may be the exciting cause, bringing about such lack of vitality as to permit a latent hereditary factor to come into play.

Other sequels include pleuritic adhesions; the development of purpura hæmorrhagica, which may occur either in the later stages of strangles or during convalescence; synovitis and a chronic lameness.

Treatment. — Any animal affected with strangles or catarrh of the upper respiratory tract should be isolated as soon as possible, and the stall or box from which it is taken should be thoroughly disinfected and left vacant if possible for three or four weeks. Disinfection of the watering pails, bits and bridles, sponges, etc., should also be carried out, while the soiled litter should be burned if possible. The place to which the patient is removed should be roomy, and there should be an ample supply of good fresh air; failing a well-ventilated box, affected horses would be better in an open shed, if they are properly clothed with rugs and bandages, and provided with a deep bedding of straw. Affected animals should not be tied up by the head but allowed to move freely in their stable wherever possible. Medicated inhalations with steam, eucalyptus, terebene or creosote afford relief and assist the removal of the nasal discharges. The nostrils must be frequently cleansed in order to avoid blistering with the discharge. The maturing of the abscesses should be encouraged by the application of hot dry packs, or of bran and linseed poultices, or by the use of a stimulating liniment or a mild blister. After a few days a soft fluctuating spot will be found and the abscess should then be opened and evacuated. It is a bad policy, however, to lance too soon owing to the danger of wounding the vessels of the part, and also the danger of setting up a more or less chronic fistula of the lymphatics. On the other hand, if the opening of the abscess is too long delayed, there is far greater destruction of tissue causing protracted healing, and there is also a danger of them rupturing internally. After being opened, the abscess cavity should be irrigated daily with disinfectants such as iodine solution 1 per cent, carbolic acid or creolin 2 per cent. The wound should not be allowed to close until all discharges cease, and it may be kept open by means of a plug of tow or cotton-wool. Any irregular abscesses must be treated as they arise, the same principles being adopted.

Medicinally very simple measures are required. Amongst the best is the administration of sulphate of magnesia ℥ii. and potassium nitrate ʒii. daily in drinking water. Chlorate of potash is also useful. In acute febrile cases the administration of salicylates ʒii. once or twice daily may be recommended. Potassium iodide is also useful, particularly in the convalescent stage, as it assists in the reduction of glands and is thought to reduce the danger of roaring or whistling as a sequel. In severe cases the hypodermic injection of nuclein in ℥ss. doses daily or on alternate days may assist recovery, while the intravenous or subcutaneous injection of 1 per cent sol. of collargol ℥i.-iss. repeated for several days has been recommended by Dieckerhoff.

The use of antistreptococcic serum has frequently been recommended. If given in very large doses it reduces the severity of the abscesses and may thus reduce the complications and unfavourable sequels. It apparently, however, has no marked effect in reducing the course of the disease. The same may be said of the use of vaccines composed of dead cultures of streptococci.

The feeding of the patient is of some importance; the food should be soft and easily digested, and wherever there is any obstruction to swallowing solid foods should be entirely withheld. In that case thin oatmeal or linseed gruels or milk should be given in a pail either standing on the ground or fixed in some low position. If the animal can swallow easily, green foods should be offered if available, or alternatively a little steamed hay, crushed oats, a linseed mash, or an occasional sliced carrot or turnip.

After strangles a long period of convalescence extending over a month or six weeks at least is necessary after apparent recovery, and then the horse must be submitted to a full course of conditioning before being put to work. Another alternative is to turn the animal out to grass, if the weather is suitable, for a couple of months.

G. H. W.

EQUINE INFLUENZA

Epizoötic Cellulitis; Pink-eye

Equine influenza is a highly contagious fever of equines, characterized by derangement of the nervous and vascular systems and catarrh of the mucous membranes. There is no connection between it and any disease of other species.

Etiology.—The organism causing it has not been satisfactorily demonstrated, though numerous organisms have from time to time been regarded as the cause. From the various manifestations of the disease the writer believes that it will ultimately prove to be a septicæmia due to an ultra-microscopic organism, and that the local manifestations are due to invasion by secondary organisms.

It is spread mainly by infective horses, and horses which appear to have recovered may remain carriers. There may also be mediate contagion, such as infected stables and mangers, drinking utensils, nose-bags, etc. Influenza appears to be one of several diseases likely to occur amongst young horses newly drafted from the country into towns, and from which dealers' stables appear to be rarely free.

Clinically, two forms may be recognised, the **benign form** and the **malignant form** or **pink-eye.** In benign cases the course is from six to twelve days, but the severity largely depends on the hygienic conditions and the condition of the animal when attacked. In the malignant form death generally supervenes within seven days, but sometimes it produces a typhoid or low-fever form which lasts a long time.

The period of incubation is two to five days.

Symptoms.—There is no hard and fast line of demarcation between the benign and malignant forms of influenza. They differ only in the severity of the attacks and the consequent mortality. The condition commences with very great depression affecting the whole body, a cough, anorexia, and temperature elevated several degrees. Swellings appear in the pendent parts of the body—legs, muzzle, and under the abdomen—due to œdema, and this is the condition which has given rise to the term "epizoötic cellulitis," a misnomer implying inflammation of the connective tissue. The pulse is soft and slow, lacking tone, frequency normal. There is a tendency to catarrh from both nostrils and from the eyes, and the eyelids may become swollen and suffused. In the majority of cases the conjunctiva is dirty-looking with a slight yellowish tinge, due to portal congestion. In very severe cases, if the eyelids are everted and the catarrhal exudate wiped off, the conjunctiva is seen to be brightly injected, hence the name "pink-eye." In this form the eyeball itself may be involved and ophthalmia set up, resulting in permanent blindness.

The catarrh may extend to the lungs, producing broncho-pneumonia with increased coughing and blowing, and the physical changes can be detected by auscultation and percussion.

In other cases the catarrh extends to the alimentary tract, producing superficial enteritis and diarrhœa, but the disease may exist without affecting either the lungs or alimentary tract. In the intestinal form the catarrh may extend up to the bile ducts, giving rise to **biliary fever.** In this case you get suddenly appearing jaundice requiring urgent treatment. Jaundice may also come on late in the course of the disease when the patient has apparently been doing well. The conjunctiva becomes suddenly very yellow and the temperature goes up acutely and may cause a relapse.

In general in influenza the temperature does not rise after the first symptoms have been seen, it gradually declines. An undulating temperature in influenza is not a favourable sign.

In some apparently uncomplicated cases there is very great weakness, most noticeable in the hind legs, which may give rise to a suspicion of kidney affection or injured back.

Careful observation of the flanks of the horse day by day before disturbing the patient assists considerably in forming an opinion as to the progress of the attack. If the case is not doing well the flank gets tucked up, and the ridge of the asternal cartilages is quite obvious, and it is the slight modifications in the degree of this that one looks for before otherwise disturbing the patient. In many London commercial stables the horse foreman has become quite adept in gauging this symptom, and it is no unusual thing for him to tell the veterinary surgeon how the horse is progressing, and adding either "He is a bit more tucked up to-day, sir," or "He's letting his belly down a bit to-day, sir," in cases where the average layman would not observe any difference.

In some cases painful swellings suddenly occur involving the sheaths of the flexor tendons. These may be metastatic, and disappear almost as suddenly, reappearing in other legs. In other cases they become chronic, a form of tendo-vaginitis and tendonitis being set up which may even lead to contracted tendons and chronic knuckling of the fetlock joints.

Other possible sequels are roaring and purpura hæmorrhagica, but in no case does the severity of the attack of influenza afford any indication of the probability or otherwise of the sequels.

Treatment.—*Prevention.*—Practise rigid isolation of all cases of catarrhal fever. New horses introduced should be put into a separate stable for two or three weeks, especially when coming from a dealer's stable, or from a journey in a railway horse-box.

Curative Treatment.—Nursing is more important than medicines. Apply rugs and bandages, and hand rub the legs. Provide good hygienic conditions; too much fresh air cannot be provided. Give small quantities of different kinds of food, boiled oats, bran mash, fresh-cut grass, steamed hay, hay tea, carrots, turnips. Tempt the horse by offering food little at a time and often.

Medicated inhalations, or steaming the head, afford relief and assist the removal of catarrh. Mustard may be applied to the throat. If bronchitis or broncho-pneumonia has set in, then the application of mustard or a mild blister to the chest wall may be of service in the less well-bred horses, but they must be used with caution in thin-skinned animals. Some practitioners always blister a fairly large area along the sides about trace high as a tactical precaution against the animal being sent to work too soon rather than in any belief in its therapeutic value.

Internally: Hypodermic injections of nuclein ℥ss. daily. Epsom salts ℥ii.-iv. in drinking water daily. Aspirin and squills in electuary are very good; or

℞ Ammon. carbonatis } āā ʒii.
Potassii iodid. }
Quinini sulph. ʒi.
Mass. communis q.s.
Misce et ft. bolus. Sig.—One to be given twice daily.

Nux vomica ʒi. may be added to the above with advantage. The writer strongly dislikes drenching in catarrhal cases and never resorts to it if it can be avoided.

In the biliary form the following may be given:

℞ Calomel ʒi.-ii.
Pulv. opii gr. xx. to gr. xxx.
Ft. bolus.

Follow next day with

℞ Mag. sulph. ℥viii.
P. Zingib. ℥ss.
Ft. Pulv. To be administered in the form of a draught.

The eyes should be bathed with some mild antiseptic, and if the cornea is ulcerated the following eye-lotion may be used several times daily:

℞ Zinci sulph. gr. iii.
Tinct. opii aquosæ ʒi.
Aq. destil. ad ℥i.
Misce et ft. collyrium.

For opacity of the cornea blow in a little hydrargyri subchloridum once daily.

Although various sera and vaccines have been recommended the writer does not know of any likely to prove serviceable, especially as the cause is not yet known.

The period of convalescence must be a long one and the horse should not be worked for at least a fortnight after he appears well. Then he should have a course of conditioning. If worked too soon, pneumonia may result, and is often fatal. During the convalescent stage a course of vegetable tonics should be given. Thoroughly disinfect the stable, the manger, bits and bridles, and destroy sponges.

Differential Diagnosis.—There appear to be two diseases occurring under similar conditions as influenza which may be mistaken for it, or may even occur simultaneously with it, viz. (*a*) a catarrhal fever, which is usually associated with a profuse nasal discharge rich in organisms indistinguishable from the streptococcus of strangles, and (*b*) contagious equine pleuro-pneumonia.

The former appears to the writer more like influenza than the latter; the symptoms of nasal catarrh, cough, weak pulse, and pallid, dirty-looking conjunctiva being very much as in influenza. The loss of appetite, however, is not so complete, and the general depression is rarely so intense, while the nasal discharge is more copious and the cough usually more severe. There is also a greater tendency to swollen submaxillary glands, though not with abscess formation as is usual in typical strangles. It is probable that this affection is an atypical form of strangles.

In contagious equine pleuro-pneumonia the difference is more marked. The animal is often ill for several days before attention is called to it, because the appetite remains good even though there may be an initial temperature of 106° F. The heart is strong and the pulse full and bounding, and the conjunctiva brightly injected. All these symptoms appear to be quite the opposite to influenza. Then the horse begins to blow and the appetite becomes capricious, and on auscultation acute pneumonia may be detected. Pleurisy with effusion follows. The crisis is reached from five to seven days after the onset of symptoms, whereas in influenza there is rarely any pronounced crisis, the condition being more depressant and less acute. Definite diagnosis is in this case a matter of some importance, for contagious equine pleuro-pneumonia may necessitate tapping of the chest, which must be done early to be effective; whereas this procedure is not called for in influenza. The difficulty of distinguishing is certainly considerable in those cases of influenza in which broncho-pneumonia has been set up. The history of the case may then help, since the pulmonary complication is usually a later stage of the disease in influenza. Some continental observers regard contagious equine pleuro-pneumonia as a pectoral form of influenza, but to the writer the pronounced differences in most of the symptoms strongly support the view that it is an entirely different disease.

G. H. W.

CONTAGIOUS EQUINE PLEURO-PNEUMONIA

Contagious Gangrenous Pneumonia of Horses

Contagious pleuro-pneumonia of horses, which may also affect asses and mules, is a rather commonly occurring affection, characterized by high fever and the early onset of pneumonia, which is frequently followed by necrosis and gangrene of the lung and also frequently by a putrid pleurisy. By many authorities this disease is regarded as a pectoral form of influenza, but differences are usually so well marked as to justify the conclusion that it is an entirely different affection. In other cases, however, there are points of resemblance, especially in those forms of influenza complicated with pneumonia.

Etiology.—This disease is generally ascribed to an ovoid bacterium, with bi-polar staining of the so-called septicæmia-hæmorrhagica type, which is always found present in the lung lesions and in the pleural exudate. In many cases there is a double infection and the ovoid organism is found in association with a streptococcus frequently present in pairs (as a diplococcus), which is indistinguishable from the streptococcus of strangles. When necrosis has been set up putrefactive organisms propagate in the dead tissue, setting up gangrene. Such organisms, however, are secondary invaders. The virus apparently lives a long time outside the animal body, and stables in which affected animals have been remain infective for a considerable period and certainly longer than after influenza. Consequently, it is one of the affections frequently met with in dealers' stables and railway horse-boxes, and horses that may have been in these places for a comparatively short time may become infected and subsequently develop the disease some days after arrival on fresh premises.

The common method of infection is by inhalation, though it is probable that some cases are set up by ingestion of food materials and water contaminated by nasal discharges from affected animals.

The period of incubation is from two to seven days, occasionally a fortnight, but it is not an uncommon thing to find the disease occurring in a stable two or three weeks after an affected animal has been removed.

Lesions.—The common lesion consists of pneumonia of the lobar type with an extensive firm hepatization. This is followed by necrosis and gangrene in which the affected tissue breaks down very readily. On cutting the affected lung the exposed surface is granular and of a greyish-brown or greyish-yellow colour, and frequently a number of small necrotic areas containing greyish-brown or greenish broken-down tissue. The necrotic areas may vary in size from that of a pea to the size of a man's fist or larger. The bronchial tubes contain an offensive brownish purulent exudate.

When the pleural sac is involved the condition is usually bilateral and a turbid exudate is present in the chest. The pleura is covered with a thick fibrinous exudate, yellow in colour. The amount of exudate may be very copious, even up to several gallons, in each side of the chest and causes collapse of the lower parts of the lungs.

Symptoms.—The animal is generally ill for a day or two before attention is called to it, mainly on account of the fact that the appetite remains good, a condition which is very unusual in the horse in other acute affections. The only thing that may have been observed is that the animal does not work so well, is not so free in gait, and has an occasional cough. On taking the temperature in such a case it is frequently found to be 105°-106° F. If the temperatures of other horses in the stud are taken it will often be found that unsuspected horses have a similar high temperature. If horses in this condition are put to work the disease develops very rapidly and proves fatal in a large proportion of cases. On the other hand, when a diagnosis is made in the early stages and the animal rested at once the affection may run a much milder course. After the first few days the appetite is lost or is very capricious, the patient becomes somewhat depressed, the respirations are increased and blowing in character; the pulse is generally strong, full, and bounding in the early stages—a marked contrast with the soft, slow, feeble pulse so common in the early stages of catarrhal influenza. In advanced fatal cases, however, the pulse becomes soft, weak, and irregular. The mucous membranes are injected and bright red, sometimes with a yellowish tinge (brick red), while there is often a slight discharge from the eyes and a swelling of the eyelids. There may be a nasal discharge, but this is by no means constant, particularly in the early stages. When present, it varies in quantity and character. It usually commences by being thin and watery and not very copious, but in the severe cases it becomes thicker and more copious and of a dirty brown colour, sometimes blood-stained. Coughing is also variable. It is, however, usually present to some degree throughout the course and becomes more pronounced and painful when pleurisy develops. At the same time there is considerable pain in the chest wall, and the patient frequently grunts, especially if made to move round the box. This painful condition subsides as the pleurisy advances and exudation into the chest occurs. With the temporary relief of pain the patient may become a little brighter and pick a little food, but respiration usually becomes more difficult owing to the fluid in the chest. Œdema is also likely to occur under the chest and the fore-limbs. There is usually some tendency to constipation.

On auscultation there is a loss of sound in extensive areas of the lung owing to consolidation, while increased vesicular murmurs may be observed in the non-inflamed areas. Increased bronchial sounds may also be heard. When the pleura is affected in the early stages friction sounds are present, but when exudation occurs friction sounds are lost. All respiratory sounds in the lower part of the chest are lost and movement of the liquid in the chest may be detected. In cases where there is some doubt as to the presence of fluid in the chest an exploratory puncture under sterile precautions should be made. There is no objection whatever to this course, and undoubtedly many valuable horses are lost through lack of an early diagnosis of fluid in the chest and the consequent affording of relief.

The crisis is usually reached between the fifth and seventh days. From this point in favourable cases the temperature declines gradually, the patient becomes somewhat brighter and picks food, while the secretions and excretions slowly improve.

In unfavourable cases **gangrene of the lung** supervenes. This is ushered in by a sudden fall of temperature almost to normal, a sudden return of the appetite and brighter appearance of the patient. This is so pronounced that one must carefully guard against being misled, though the deception is not likely to last long. During the next day the breath and nasal discharge become fœtid, the patient becomes very depressed, and the case terminates fatally in from twenty-four to forty-eight hours.

In all these cases there is a very marked loss of condition and affected animals rarely lie down. When they do so it is usually regarded as an unfavourable sign.

Prognosis.—Contagious equine pleuro-pneumonia is an extremely serious disease and the mortality depends largely on the rapidity with which the illness is recognised and the animal taken from work and placed under favourable conditions. The fact that diagnosis of the first cases is usually delayed is responsible for the deaths of a large proportion of them and probably 50 per cent of those cases die. The prognosis is more favourable, however, in subsequent cases, and if the temperatures of all the remaining animals of the stud are taken daily and the horses put under treatment immediately there is a rise of temperature, recovery takes place in a large majority of them. This is due to the fact that the pneumonia is then often very slight and pleurisy does not develop. The prognosis is also unfavourable in those cases where there is a sudden fall of temperature indicating the onset of gangrene.

Treatment.—Rigid isolation of the first case should be practised. The patient should be placed in a box with an ample supply of good fresh air. There is no objection to the horse being left in the open so long as he is protected against the elements. The body surface should be kept warm with rugs and bandages. Medicated inhalations should be resorted to and the patient tempted with such foods as a little steamed hay, a little freshly-cut grass or clover, boiled oats, linseed mash, hay tea, gruel, or milk. Good hygienic conditions and nursing are of far greater importance than medicinal agents. Medicines, however, may be found of considerable service in improving the vitality and general resistance of the patient. Drenching should be avoided owing to the danger of producing "drenching pneumonia" and gangrene.

The same medicinal agents may be administered as in the treatment of equine influenza (see pp. 22-23).

Miessner has reported excellent results from the hypodermic or intravenous injection of 5 grammes of neosalvarsan dissolved in 100 c.c. of water. The solution should be freshly prepared and injected as early as possible in the case. Miessner claims that it produces a rapid fall in the temperature and increased appetite. The pneumonic changes are checked and an early cure follows, with absence of unfavourable sequelæ.

In spite of the fact that constipation may exist purgatives should be avoided owing to the danger of setting up super-purgation and on account of their great cardiac depressant effect. Counter-irritants applied to the chest wall afford relief. These may consist of the application of hot blankets or cold blankets covered with a mackintosh sheet, or in the case of thicker-skinned horses the application of mustard plaster or a mild cantharides blister. The hypodermic injection of nuclein ℥ss. daily is often useful.

When pleurisy exists the chest should be tapped *as soon as possible.* This should be done about the eighth intercostal space with all sterile precautions. It should be carried out at the earliest moment when there is any fluid at all in the chest and may be repeated every other day.

When the breath is offensive the intratracheal injection of creosote ʒss. in glycerine ℥ss. may be resorted to and is said to give good results. It is doubtful, however, if anything is of service when gangrene of the lungs has been set up.

The use of various vaccines and so-called phylacogens prepared from either the ovoid organisms or streptococci have been recommended, but they are by no means specific in their action and are likely to give rise to disappointments.

A long period of convalescence is essential. Animals put to work too soon are very liable to contract a relapse which is almost always fatal. After pneumonia very thorough disinfection of the stable must be practised and it must be left vacant for at least a month.

G. H. W.

PURPURA HÆMORRHAGICA IN THE HORSE

Petechial Fever ; Anasarcous Fever ; Hæmorrhagic Toxæmia

Purpura hæmorrhagica is an acute or subacute toxæmic affection, usually secondary, rarely primary, characterized by small hæmorrhages into the mucous membranes, skin, and internal organs, and œdematous swellings of the subcutaneous connective tissues particularly of the dependent parts. Some English writers (Williams and Robertson) in former times have described the condition as "scarlatina," but as there is not any resemblance to or association with that disease in the human subject, nor is it transmissible to man, the name is open to very great objection.

Etiology.—The true exciting cause of this affection of the horse is unknown. It is often said to be a microbic affection, and an infective disease; by some it is said to be a septicæmia due either to an ultra-visible virus or to a streptococcus or a staphylococcus; by others it is said to be due to a bacillus or a pasteurella. It may be that the local lesions are caused by a plugging of some of the capillaries with organisms.

Against the theory of specific microbic origin is the fact that no definite micro-organism has yet been isolated capable of producing the affection, and, moreover, transfusions of blood from affected to healthy horses and inoculations of discharges from affected animals have all been uniformly negative. Although purpura is said to occur occasionally as a primary affection, it is far more frequently a sequel to or a complication of some well-marked debilitating disease, such as strangles and influenza, while cases are recorded following various infected wounds, chronic abscesses, and suppurating lymphatic glands. Dieckerhoff, whose views on purpura are especially worthy of consideration, regards the disease as an auto-intoxication arising from various primary foci, such as suppurative catarrh, suppurating lymphatic glands, etc., due to micrococci and causing irritation or other injury to the vessel walls, and thus leading to hæmorrhages. Cadeac considers it to be due to the absorption of any bacterial toxin having a vaso-dilatory action.

It is probable that the reported primary cases of purpura are really secondary, but that the primary condition may have escaped observation, for the mildness or severity of the primary affection, even when observed, appears to bear no indication of the probability or otherwise of an attack of purpura. An animal may have a comparatively mild attack of strangles or influenza, and appear to be doing quite well, and suddenly develop purpura hæmorrhagica; while other horses in the same stables may have a very severe attack of the primary affection and yet make an uneventful and uncomplicated recovery. In such stables there may be a single case of petechial fever, or there may be several, but one is quite unable to find any causes operating in the affected that are not also present in those that escape the unfortunate sequel.

Clinically, the disease is often associated with bad hygienic conditions such as dirty stables, badly ventilated and badly drained, and with bad flooring, and sewage-contaminated water. By many it is thought to be directly attributable to such conditions. But purpura hæmorrhagica may and does occur in ideal stables where none of these bad conditions obtain, and again debilitating diseases as strangles and influenza are quite common in the worst stables without purpura occurring as a sequel. Such conditions, then, can only be regarded as contributory inasmuch as they tend to reduce the vitality of an animal and militate against his making a good and complete recovery from his primary complaint.

The condition, then, would appear to be due to an exhausted state of the animal's vitality through previous disease, producing alterations in the constitution of the blood, such as reduction of the fibrin-forming elements, and this together with great loss of tone of the heart and the capillaries favours hæmorrhages and transudations.

Symptoms.—Apart from the common history of previous disease from which the patient may appear to be making a good recovery, the special symptoms of purpura hæmorrhagica are usually developed rather suddenly, and may be well marked when first observed; sometimes they appear gradually and in succession. The most characteristic features consist of petechial markings on the visible mucous membranes, and œdematous swellings of dependent parts. The petechiæ may be seen best on the nasal mucous membranes, the conjunctiva over the membrana nictitans, and inside the lips. If the latter are completely everted the ecchymoses are generally very obvious. In the mare they may also be seen on the mucous membrane of the vulva. They consist of blood spots varying in size from that of a split pea to the size of half-a-crown, or larger where several have coalesced. A nasal discharge may accompany them and may be slightly blood-tinged. They occur suddenly and may disappear as suddenly, sometimes reappearing.

The skin lesions in some cases commence simultaneously with the petechiæ, and consist of more or less numerous flat circumscribed urticaria-like swellings from one to two inches in diameter and occurring on any part of the body. The swellings may disappear in a day or two or become confluent and more persistent. More commonly, however, diffuse swellings develop affecting principally the dependent parts; the muzzle is usually one of the first seats, then one or more limbs, the eyelids, and under the breast and abdomen. The swellings are cold and painless, and easily pit on digital pressure. The upper margin on the limbs often has a sharp line of demarcation, suggestive of the effect that would be produced by tying a cord round the upper part of the limb. A similar sharp demarcation is present at the anterior margin of abdominal swellings. At the muzzle the swellings, beginning at the nostril and lips, extend upwards and then usually end abruptly, and if a headstall is on, its weight, even if loosely fitting, may produce a groove in the swelling. A curious feature in some cases is the sudden disappearance of a swelling in one region, and at the same time the appearance of one in another place. In many cases there exudes on to the surface of the skin droplets of a sero-sanguineous fluid with no tendency to coagulate, and in horses with a light skin, as greys or creams, petechial markings may be seen.

There is usually considerable depression, the head hangs down and increases the œdema; the pulse is frequent, soft, feeble, and sometimes irregular. The internal temperature is rarely very high, it usually oscillates between 102° and 104° F. The appetite, which was probably improving on his apparent recovery from his primary affection, again fails, and this may be further intensified by the swollen lips interfering with prehension. The swelling of the nostrils may cause marked dyspnœa, and even asphyxia in exaggerated cases if tracheotomy is not performed. The swelling of the limbs interferes with flexion of the joints, causing difficulty in moving about, sometimes giving rise to fissures about the knees, hocks, and pasterns. The œdema under the body and elsewhere may so interfere with the local cutaneous circulation as to cause large sloughs of the skin and the development of large open wounds, which may become infected and lead to serious complications. The urine is often rather scanty and albuminous, and in the male micturition may be difficult from a swollen sheath or pendulous penis (paraphimosis).

Other systemic symptoms depend on what internal organs are implicated. One of the worst complications is a suddenly occurring œdema of the lungs, which will probably prove fatal from asphyxia. Pneumonia may occur as the result of saliva, food, or even drenches passing down the trachea. Such cases are usually septic and fatal. The alimentary tract may also be involved, and enteritis produced with diarrhœa and colicky pains. Such cases are also very unfavourable and usually terminate fatally. Nervous symptoms may occur, and vary from paraplegia when the spinal cord is affected, to fits and convulsions or coma when the meninges and brain are involved. Such cases are always fatal.

Prognosis is very difficult, and should be guarded. The affection is fatal in about 50 per cent of cases. Sometimes all the characteristic symptoms will disappear in twenty-four hours and the horse make a complete recovery. In other cases, when all appears to be going well the patient suddenly becomes worse and dies. The best indications are the *gradual* improvement in heart and general circulation, the gradual return of the appetite and reduction of the swellings, the absence of marked dyspnœa and of the colicky pains. In the more favourable cases a long period of convalescence—a month or six weeks is absolutely essential to a good recovery, and it is here that one is frequently handicapped by the owner, who thinks that a horse that has returned to his appetite a few days or a week is necessarily quite fit to go back to work. If a horse is sent to work too soon he is very likely to get a relapse of some kind, either pulmonary or intestinal, and will probably die.

In fatal cases death may be due to a variety of causes, viz. œdema of the brain, asphyxia from œdema of the lungs or from the swollen nose, pneumonia, enteritis, exhaustion, or from septic infection of sloughs of the skin.

Treatment.—In the first place provide the best possible hygienic conditions, a nice warm and well-drained loose-box, with plenty of fresh air.

Careful nursing and judicious feeding are of the highest importance. Foods should be easily digestible and of a laxative or demulcent nature. They should be offered in small quantities, and frequently changed. Amongst the best are linseed mashes well boiled, oatmeal gruel, eggs and milk, hay-tea and barley water; a little green meat if available, and a little steamed meadow hay with a good aroma. To all or any of these may be added a little extract of malt.

Of medicinal agents, stimulants may be useful, such as brandy, ether, or port wine, but the chief objection to them is that they will have to be given as a drench, and I do not approve of drenching at all if it can possibly be avoided. The same applies to the old-fashioned and well-spoken of remedy Ol. tereb. ℥ii. in a pint of milk given twice daily. Potassium chlorate is another of the old remedies, and it can be given in the drinking-water. Each of these agents is highly spoken of by some practitioners, and just as highly condemned by others. Liq. ferri. perchlor. in doses of ʒss. or ʒi. b.i.d. is often recommended and is useful. Adrenalin is given sometimes hypodermically, in doses of ʒi. of the 1-1000 sol. It is an excellent cardiac stimulant and may also check the hæmorrhages and œdema. Another much-favoured line of treatment consists in the intratracheal injection of Lugol's solution of iodine as recommended by Dieckerhoff. The solution consists of

Iodine 1 part.
Potassium iodide 5 parts.
Water 100 parts.

The dose is from ʒss. to ʒii. twice daily for the first two or three days, and once daily for several days afterwards. For administration the head must be raised and the strong sterile needle inserted between the rings of the trachea. The fluid must be injected very slowly indeed, and then should not cause a fit of coughing. Combined with this, if it is possible to ball the horse, and he can swallow fairly easily, I recommend potass. iodid. ʒii., acid. aceto-salicyl. and pulv. nucis vom. āā ʒi. twice daily in bolus, or in electuary. Fresh nuclein in ℥ss. doses daily hypodermically is very good. It increases leucocytes in number and vigour.

Collargol has been recommended very strongly, but it does not appear to have answered as well as anticipated. It is given intravenously at the jugular in doses of gr. vi.-xii. in 1 per cent solution in sterile water.

Various vaccines and sera have been used, but it is doubtful if these are any good, as the specific organism—if there is one—has not yet been isolated. Polyvalent antistreptococcus and antistaphylococcus vaccines and sera are the kind used.

Local Treatment.—This should consist of the application of mild astringents such as lead lotion or white lotion. The swellings must not be scarified to relieve œdema, as that only increases the probability of sloughs If sloughs have occurred the wounds should be dressed with ordinary antiseptic emollient and protective dressings. Moreover, one must not attempt to reduce the swellings with pressure bandages. Use bandages for warmth if necessary, but let them be quite loosely applied. Otherwise they are better not used at all. If there is paraphimosis the penis should be supported by means of a suspensory bandage. If the nasal swellings threaten to asphyxiate then tracheotomy may be performed, but this should not be done too hurriedly owing to the great dangers from infection in this condition.

On recovery a long period of convalescence is necessary, during which the patient should have light exercise, a generous diet, and a course of tonics.

Purpura Hæmorrhagica in Cattle

This disease has been recorded in cattle in various parts of the world, giving rise to symptoms somewhat resembling those in horses and of similar obscure origin. It is probable that some of the cases were septicæmia hæmorrhagica.

The *symptoms* recorded include anorexia and suspended rumination and lactation, general stiffness and debility, hæmorrhages and swellings in the skin and petechial markings of the mucous membranes, often accompanied by blood-stained discharges from the nose and vulva, and sometimes with the fæces. Great emaciation and anæmia follow. Recovery occurred in the majority of cases recorded in from three to six weeks, but in others death followed from septicæmia or intestinal hæmorrhage.

Treatment.—The patient should be placed under the best hygienic conditions possible, and the appetite coaxed with green foods and palatable concentrated foods such as linseed cake, crushed oats, etc. The administration of salines and diuretics is usually effective. A dose of sulphate of magnesia or sulphate of soda may be followed by salicylates and potassium nitrate or potassium iodide as in the case of the disease in horses. Turpentine, by the mouth, intratracheal injections of iodine solution, and hypodermic injection of collargol and of adrenalin have also been recommended. French writers strongly advise bleeding in the early stages, but it is rather contra-indicated by the already debilitated condition of the patient.

Local treatment should be adopted as for the horse.

It is doubtful if purpura hæmorrhagica occurs in cattle in the British Isles. The writer has frequently met with cases of anasarca of the limbs and under the sternum and abdomen which might possibly have some resemblance to the equine affection, but they have been unaccompanied by the petechial markings on the visible mucous membranes—typical of the condition in the horse. He has also found it more amenable to treatment and has usually regarded the condition as due to local causes.

Purpura Hæmorrhagica in Dogs

Several cases resembling purpura hæmorrhagica have been described in dogs, in which the principal symptoms consisted of emaciation and great debility associated with the appearance of small hæmorrhages in the skin and mucous membranes, and the internal organs. The heart was very weak, and dropsical swellings of the limbs and body cavities occurred. There was an absence of febrile symptoms, and urine and fæces were blood-stained.

All the recorded cases terminated fatally.

G. H. W

GLANDERS

Fr. **Morve**—*Ger.* **Rotz**

Glanders is an infectious disease of solipeds (horses, asses, and mules) and is communicable to man, numerous cases of human infection having occurred as a result of the handling of infected animals and also in laboratory work.

The carnivora are also susceptible to infection and the disease has frequently been met with affecting the wild carnivora in zoological gardens, as a result probably of their having been fed with raw infected horse-flesh. Cats, dogs, and goats are susceptible, sheep less so, and cattle are immune. Two clinical types are recognized depending upon whether the lesions are present (*a*) in the internal organs, *glanders*, or (*b*) in the skin and subjacent lymphatic tissue, *farcy*. The former is the type more commonly met with.

Bacteriology.—The causal organism, *B. mallei*, was discovered by Löffler and Schütz in 1882 and was later described by Kitt, Weichselbaum, and others. It is a short, usually straight rod with rounded ends, which occurs as a single element, occasionally in pairs or short chains. It is from 3 to 6 microns in length by from ·25 to ·4 of a micron in breadth, non-motile and does not sporulate.

The organism is an obligatory parasite and sporadic cases of the disease therefore do not occur. In the body it is essentially a tissue parasite with a predilection for the lymphatic system and is recoverable from pus, discharge from ulcers and the nasal mucous membrane, and from infected tissue. It stains with watery solutions of the aniline dyes, especially those which contain a mordant such as Löffler's alkaline methylene-blue, Kuhne's blue, carbol fuchsin or carbol thionin, but not by Gram's method. It grows readily under aerobic conditions in all the artificial media commonly employed, especially those to which glycerine or serum has been added. Upon potato a characteristic honey-like growth appears within 48 hours. During the next two to three days it becomes brown and later chocolate-coloured, and the surrounding potato is blackened.

Experimentally most of the small laboratory animals are susceptible, especially the guinea-pig. In male guinea-pigs a glanders sarcocele usually results in from three to ten days after the intraperitoneal injection of pure cultures of the organism if the dose is not sufficiently large to produce a rapidly fatal result.

Lesions.—In the horse, the commonest lesions are pulmonary nodules, after that enlargement and induration of the submaxillary bronchial and tracheal lymphatic glands, ulceration of the nasal septum, farcy lesions, and rarely lesions of the abdominal organs (spleen, liver, glands, kidneys, intestines, testicles, and uterus) and sometimes of the bones. Strips of infected lung on palpation feel gritty or as though studded with small shot. The pulmonary nodules vary in size from a millet seed to a pea and on section are greyish white in colour in the centre surrounded by a hyperæmic zone. In older nodules this zone is replaced by fibrous tissue, and the central area becomes encapsulated. Microscopically a young nodule is found to be made up of a central area and three distinct zones. The central area is composed of alveoli filled with polymorphonuclear leucocytes. The contents of this area undergo a peculiar necrosis which is characterized by disintegration of the cell nuclei, the resulting particles of which, however, retain their affinity for nuclear stains (*Chromatotaxis*). The three zones consist of an inner or epithelioid ring, an intermediate zone of swollen parenchymatous cells, and an outer zone composed of alveoli containing fibrinous exudate. Old nodules consist of a granular central area surrounded by a fibrous capsule.

Nasal lesions, although usually present in advanced cases of glanders, are of rare occurrence compared with the pulmonary lesions. They originate as nodules in the mucosa of the nasal septum, which, as a result of their disintegration, give rise to characteristic ulcers. In the parenchymatous nodules the disintegration is characterized by a cloudiness which progresses from the centre outwards, and during the process hyperæmia and cellular infiltration occur in the surrounding tissue. Nasal ulcers are most frequently observed upon the lower part of the septum, but in cases of long standing they may occur upon any part of its surface and also upon the turbinate bones. In such cases the frontal sinus is also not infrequently found to be affected and to contain pus rich in glanders bacilli. Perforation of the septum has frequently been observed, and in cases of such long standing, stellate cicatrices representing healed ulcers are also not infrequently found. The submaxillary lymphatic glands are very commonly found to be affected; they become enlarged, usually indurated, and in many cases adherent to the jaw-bone. Upon section the lobulation of the gland is found to be remarkably pronounced. The gland substance shows but very little congestion, and small nodules may be present in the matrix. Infection of the glands may occur without the coexistence of nasal lesions, but when ulcers are present the glands are almost invariably found to be affected. In acute cases of glanders accompanied by nasal ulceration there is sometimes little or no induration of the submaxillary glands, which are enlarged and soft, but very seldom burst spontaneously.

Farcy Lesions.—Farcy is characterized by a diffuse subacute or chronic lymphangitis accompanied by lymphadenitis which may occur on any part of the body, but is usually found affecting a hind, rarely a fore, limb. There is generally some enlargement of the limb extending from the fetlock to the hock or a little above it.

Farcy "buds" are small subcutaneous abscesses varying in size from a pea to a hazel nut, which break down and burst, discharging a characteristic clear oily pus. They are commonly found along the course of enlarged lymphatic vessels (farcy cords).

Symptoms.—In the great majority of cases the disease is of a chronic nature. Its development is slow and insidious and is accompanied by progressive emaciation, irregularities in temperature, and polyuria. Ulceration of the mucous membrane of the nostril, particularly that covering the septum nasi, leading to the formation of a peculiar yellowish semi-transparent viscid discharge which may later assume a muco-purulent character, together with enlargement, unaccompanied by heat or tenderness, of the submaxillary lymphatic glands of one or both sides constitute the most characteristic chain of symptoms of the disease. The peculiar cold, enlarged gland is spoken of as a "jug." When the glands of one side only are affected it is almost invariably found that the visible ulceration is confined to the nostril of the same side. These symptoms may, however, be entirely absent in animals which react to mallein and which, on post-mortem examination, are found to be affected with glanders.

In farcy, which is really a subcutaneous form of the disease, the lymphatic glands and their vessels become infected. Nodules are found along the course of the lymphatics, which are themselves enlarged and resemble cords (farcy pipes) under the skin. The nodules, which frequently burst and discharge a peculiar glairy, creamy pus, are spoken of as farcy buds. Farcy lesions are most commonly seen in the hind limbs, and accompanying or even preceding them there is a general enlargement of the affected member so that in the early stages of the disease it resembles an ordinary lymphangitis. In the later stages farcy has to be distinguished from epizoötic lymphangitis, which disease it very closely resembles, and from ulcerative lymphangitis or cellulitis.

Occasionally an acute form of the disease is met with, especially in the case of asses and mules. There are then rigors, high temperature, a muco-purulent, perhaps blood-stained nasal discharge, with a fibrinous deposit upon the mucous membrane. There may be dyspnœa and stertor accompanied by marked depression. There is, in such cases, not infrequently a diffuse subcutaneous abscess formation, and death may occur in a few days. Such cases of acute glanders are often, in the horse especially, a flare-up of the chronic form of the disease.

Glanders, however, may exist in horses in good bodily condition, giving no indication that they are affected with the disease; such cases are usually not suspected and are only discovered as the result of a reaction to the mallein test which has been applied to the stud after the discovery of an outbreak.

Diagnosis.—Clinical glanders may be diagnosed with a reasonable degree of certainty from the external lesions. Microscopic examination of suspicious discharges may help to confirm the diagnosis, but the absence of the specific bacillus does not permit of a negative diagnosis.

Cultural methods may be employed if uncontaminated material is available, and a male guinea-pig may be injected intraperitoneally with pus from a freshly opened abscess.

Diagnosis by Means of Mallein.—Mallein is a glycerine extract of *B. mallei* and is prepared by growing a strain of known virulence in glycerine broth for three or four weeks. The culture is sterilized by heating at 115° C. in the autoclave or by steaming at 110° C. The bacillary bodies are then removed by filtration through a Berkefeld filter, and the limpid filtrate to which ·5 per cent of carbolic acid is added to prevent decomposition, constitutes mallein. Mallein so prepared is that commonly used in this country—the dose employed being 1 c.c. Concentrated mallein is prepared by evaporating the original culture to one-tenth of its volume in the water bath and filtering through Chardin paper. Dry mallein is prepared by adding several times its volume of alcohol to crude mallein and drying the resulting precipitate.

Treatment is not permitted. Repeated injections of mallein which really consists of vaccination, are said to promote recovery. Probably such treatment simply masks the effect of subsequent test doses.

Preventive.—All new purchases should be malleined before coming into contact with horses already in the stud. Buckets should be provided for horses at work instead of permitting the use of a common trough.

Method of applying the Mallein Test.—*The Subcutaneous Test.*—The mallein is injected into the subcutaneous connective tissue at about the middle of the side of the neck. The temperature of animals to be tested should be taken on the day previous to the injection, at the time of injection, and at the ninth, twelfth, and fifteenth hours afterwards. It is not advis-

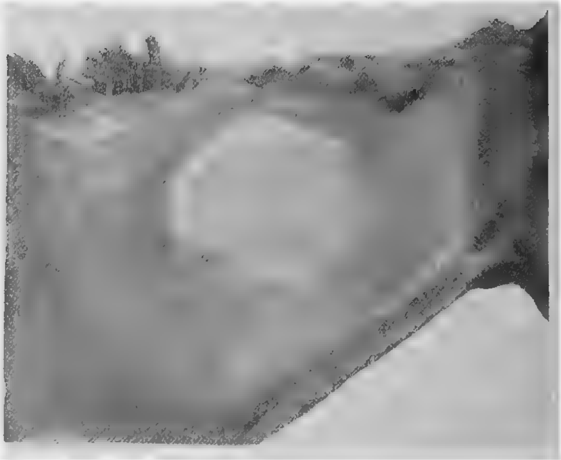

Fig. 5.—Glanders. Local reaction to the subcutaneous mallein test.

able to inject animals whose temperature is 102° or over. In the case of infected animals the temperature rises to 104° C. or more during the first fifteen hours, and at the site of inoculation there develops a large, hot, painful swelling, which disappears slowly. The maximum size (five to twelve inches in diameter) is not attained until the second day, after which it gradually subsides, although often not disappearing until from three to five days after injection. In non-glandered animals little disturbance, if any, results from an injection of mallein; the rise in temperature does not exceed 1° to 2°, and the swelling, which is rarely more than three inches in diameter, reaches its maximum size during the first fifteen hours after injection, and very rapidly disappears. In doubtful reactions where a second dose is to be employed, at least a fortnight should elapse between the first and second tests. Sometimes double the usual dose or even more is injected in subsequent tests.

The Ophthalmic Test.—This test consists of the instillation of concentrated, preferably glycerine free, mallein into the conjunctival sack, or of painting it on to the conjunctiva. Infected animals show marked congestion of the conjunctiva accompanied by swelling of the eyelids, photophobia, and the formation of a muco-purulent discharge. The test does not appear to be as reliable as the corresponding one employed for the diagnosis of tuberculosis.

Mohler and Eichhorn claim to have obtained very satisfactory results from the use of a concentrated mallein prepared by evaporating ordinary mallein to one-tenth of its original volume, adding 5 per cent phenol and 20 per cent glycerine, and again reducing to one-tenth of its original volume. The dose of such mallein is ·1 c.c.

The Intra-Dermo-Palpebral Test.—Originally described by Lanfranchi, this test consists of the intradermal injection of concentrated mallein into the lower eyelid about ¼ in. to ½ in. below the margin, and midway between the outer and inner canthi. In the case of infected animals the reaction is characterized by swelling of the eyelid, which first appears in from four to six hours of the injection and increases in size and intensity up to 48 hours, usually persisting for 72 hours and not entirely disappearing for four days. In addition there is often some infiltration of the surrounding subcutaneous tissue with partial or complete closing of the eye and a copious muco-purulent discharge from the inner canthus, accompanied by marked conjunctivitis.

The Agglutination Test.—The serum of normal horses agglutinates suspension of the glanders bacillus in dilution up to 1 in 400. The serum of glandered horses agglutinates similar suspensions in dilutions of 1 in 500 c.c. to 1 in 2000 or more. The precipitin test is also occasionally employed.

The Complement Fixation Test was first applied by Schütz and Schubert to the diagnosis of glanders. The antigen may consist of a suspension of *B. mallei* in normal saline solution or of a solution of dried bacterial bodies. Occasionally also mallein is used, but is not so satisfactory. A titre of ·1 or less may be regarded as a positive reaction, and one of ·2 as suspicious, while one of ·5 has little or no significance, as normal serum may fix complement in such quantities.

J. B. B.

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EPIZOÖTIC LYMPHANGITIS

African Farcy ; Lymphangitis Saccharomycotica

Epizoötic Lymphangitis is a chronic contagious disease characterized by inflammation and suppuration of the subcutaneous lymphatic vessels. It affects principally horses, asses, and mules, but has been reported to affect cattle in Japan. It is not inoculable to man. The disease has rather a wide distribution, occurring on the continents of Europe, Asia, and Africa. It does not exist at present in the British Isles, though it was introduced from South Africa after the Boer War, and was stamped out in the course of a year or two.

Etiology.—This disease is caused by an organism which is called the *Cryptococcus of Rivolta*, which is not a bacterium, but is a large ovoid organism of the yeast type, and which reproduces by budding. When examined microscopically the organism is seen to have a refractile capsule which gives it the appearance of having a double contour. It is best stained by the Claudius method.

Boquet and Negri claim to have cultivated the organism on a medium consisting of horse-dung agar covered with a film composed of maturated lymphatic gland pulp from a horse. After several sub-cultures the organisms could be grown on other media. With the cultures thus obtained these workers have been able to reproduce the disease experimentally in the horse.

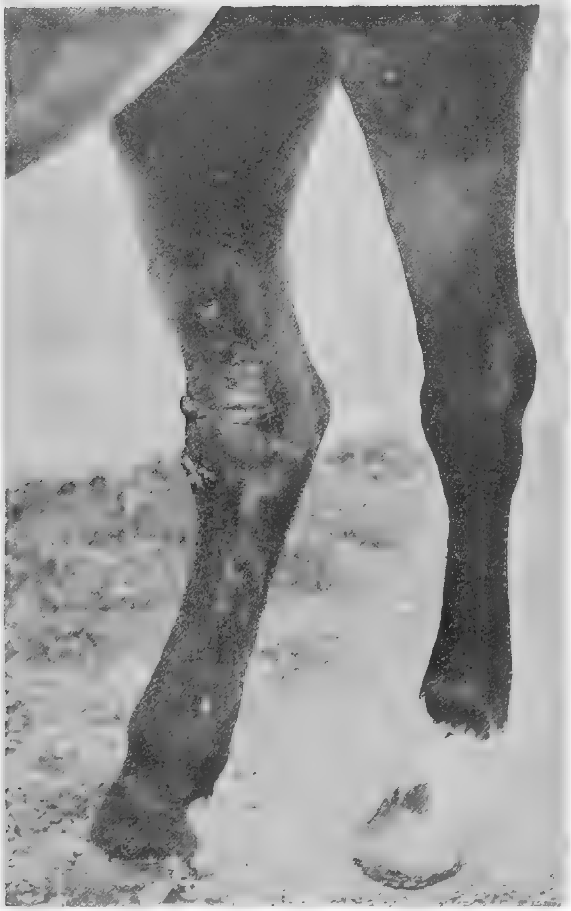

Fig. 6.—Epizoötic Lymphangitis, infected from a cracked heel. (Pallin.)

The natural method of infection is by inoculation through the skin by means of a scratch or a small wound which may have entirely disappeared before the symptoms develop. Infection may also take place through the mucous membrane, such as the conjunctiva, without any wound. The disease is never sporadic, and the cause of an outbreak is always the introduction of an affected horse. Infection may be carried by harness, grooming utensils, clothing, and infected litter. It has also been suggested that flies may be mechanical means of transference.

The period of incubation varies from two weeks to six months, and the disease runs a chronic course, and may extend from three months to a year. The mortality is about 10 per cent.

Symptoms.—Lesions most frequently commence on the limbs, of which one or more may be affected. They may also occur on the head and neck, withers, scrotum, udder, and vulva, and may extend to any adjacent mucous membrane such as the vulva, conjunctiva, and less frequently to the nasal mucous membrane. They commence as an engorgement of the lymphatic vessels, producing elongated cords leading up to the lymphatic glands of the part. On the cords of these vessels there develop small enlargements resembling glanders farcy buds, and varying from the size of a pea up to a hen's egg. They are at first firm and painless, but become painful and develop a soft pointing centre. They then burst and liberate a thick, creamy-yellow granular pus, which is often blood-streaked. The discharge subsequently becomes more sticky and yellow. After bursting the buds virtually constitute ulcers with a raised convex margin, the edges being semi inturned. When the surface is wiped, the base is red or rose-pink in colour. The buds nearest the lymphatic glands though the first to form are often the last to burst. The lymphatic glands become large and indurated and may also form suppurating centres, which may either burst or undergo resolution without bursting. The healing of the wounds is slow if left untreated. When the lymphatic glands are affected the whole limb may become considerably swollen, but it is much less painful than in the sporadic lymphangitis. When the mucous surfaces are affected the first thing to be observed is a thick mucous discharge, followed by the formation of a crater-like ulcer which develops into a small granulation. In the case of the eye, this most often occurs on the membrana nictitans or third eyelid.

There are not many systemic symptoms. In mild cases the bodily condition may be well maintained, but when the lesions are extensive the animal may become emaciated and debilitated. In such cases the temperature fluctuates between 101° and 105° F.; the patient then gets gradually worse, and in bad cases death follows from exhaustion.

Diagnosis.—The most satisfactory method of making a positive diagnosis is microscopic examination of pus from one of the lesions, and the demonstration of the cryptococci which are usually present in considerable numbers in all but cicatrising lesions undergoing resolution. The creamy character of the pus from a newly opened bud and the granulating base of the ulcers assist in distinguishing the condition from glanders. Moreover, the tendency to softening and suppuration of the lymphatic glands is greater in this condition than in glanders. Horses affected with epizoötic lymphangitis do not react to the mallein test.

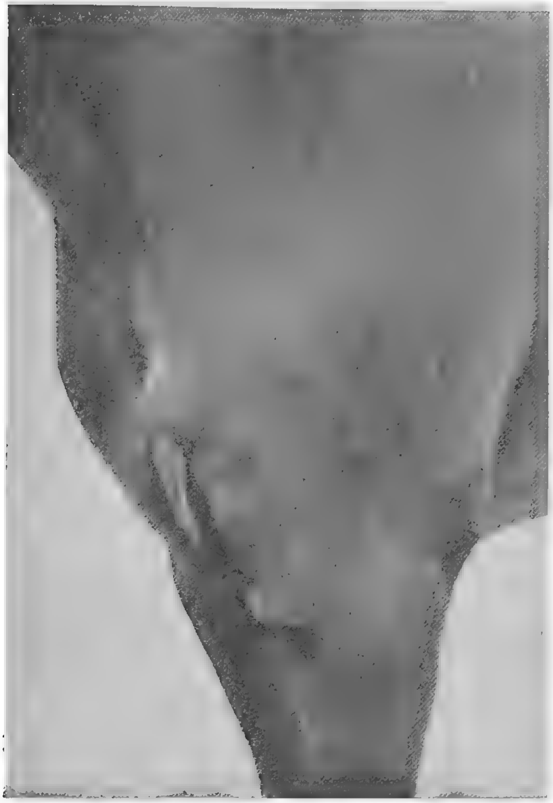

FIG. 7.—Epizoötic Lymphangitis. (Pallin.)

Lanfranchi has introduced a diagnostic test for epizoötic lymphangitis which he claims is very reliable. His testing material is prepared from pus obtained from fresh unopened buds by treating it first with twice its volume of sulphuric ether, and evaporating after twenty-four hours. It is then mixed with distilled water equal to the original volume and shaken. After twenty-four hours it is heated to 80° C. for twenty minutes in a water bath. It is subsequently centrifuged and the liquid thus separated constitutes the testing material. The dose is 3 c.c. and the injection is made into the lower eyelid. In horses not affected with epizoötic lymphangitis a slight swelling of the eyelid may occur, reaching its maximum size in eight to ten hours, and then gradually disappears within twenty-four hours. In horses affected with epizoötic lymphangitis a local swelling commences in about two hours and gradually increases up to twenty-four or forty-eight hours, involving the entire eyelid and extending down the face. The swelling is tense, hot, and painful. At the same time a yellowish pus-like discharge escapes from the inner canthus of the eye and lasts for twenty-four to forty-eight hours. There may also be a temperature reaction of several degrees commencing about the eighth hour and attaining its maximum between the eighteenth and twenty-fourth hours. It gradually falls to the normal within forty-eight hours. The reaction is said to be more pronounced in the early stages of the disease before the lesions are fully mature. It may therefore prove to be a very valuable agent in any effort to exterminate the disease since it may permit diagnosis before the animal becomes infective from discharging lesions.

The test can only be used on a large scale when some method of cultivation of the cryptococcus has been discovered, otherwise there will not be sufficient material available. It is possible that Boquet and Nègri have solved this problem (see *ante*).

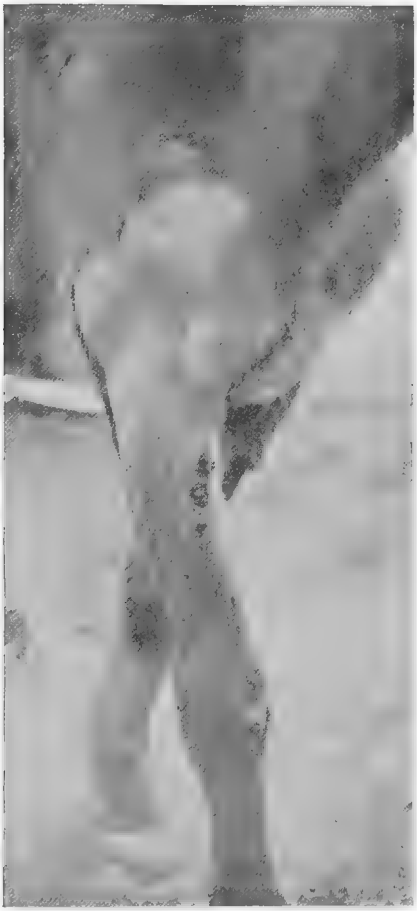

FIG. 8.—Epizoötic Lymphangitis, showing lesions up the forearm extending from the knee, the seat of infection. (Pallin.)

Prognosis.—Where energetic measures are taken early, recovery should occur in at least 90 per cent of cases. Relapses and recurrence, probably due to reinfection, are not infrequent. When the lesions are limited to the limbs or places where they can be surgically removed the cases are hopeful, but lesions in the region of the withers and of the face are amongst the worst—in the former case on account of the direction of the lymphatic vessels. In far-advanced cases recovery is not hopeful.

Treatment.—This disease is scheduled under the "Contagious Diseases of Animals Act" in the British Isles, and treatment is not permitted—affected animals being slaughtered forthwith. In countries where treatment is permitted the treatment must be somewhat drastic if it is to be of service. Wherever possible the lymphatic cord should be completely extirpated after being ligatured above and below. The wound should then be cauterized and subsequently treated under antiseptic precautions. In less extensive cases and in situations where radical operation is impossible, the ulcers should be curetted and cauterized with the hot iron, and subsequently dressed with tincture of iodine. No subsequent cauterization is necessary as it only retards the healing process. It is stated that the application of a blister of biniodide of mercury may check the progress of the condition if applied in the early stages.

Internally various remedies have been administered, including iodine, arsenic, mercury, copper sulphate, quinine, and strychnine, but they are very unsatisfactory, and local treatment is of far greater importance.

Affected animals should be isolated, and thorough disinfection of harness, utensils, and clothing should be carried out. All contaminated litter should be destroyed by burning or deep burial, since flies may transfer infection. The patient should be dressed with applications containing creosote or creolin to ward off insects.

G. H. W.

SPOROTRICHOSIS

This disease affecting horses is due to the inoculation of a sporothrix through a wound or scratch. Common seats of infection are cracked heels, fetlock wounds from "brushing," and harness excoriations.

The lesions consist of cutaneous nodules, usually multiple. There is no tendency for the nodules to coalesce, and there is little or no pain associated with them. They may be found round the pastern or fetlock, or higher up the limb (fore or hind), and occasionally on the neck, face, and trunk. The lesions commence as small circumscribed swellings which average about the size of hazel nuts. As they develop the skin over the centre becomes thin and soft owing to the formation of a small amount of pus. A little oozing takes place, matting the hair and forming a crust which becomes detached, exposing a raw surface. A little thick yellowish-white pus may be observed, but there is rarely any appreciable discharge of pus sufficient to trickle from the lesion. The raw ulcer averages about half an inch in diameter, and has a depressed centre containing a little pus, and is soon covered again with a dry hairless scab. It usually heals by granulation in 3 to 4 weeks, leaving a permanent hairless scar which gets smaller with age. Secondary lesions may appear in from one to three months, thus maintaining the affection for a considerable time even though the individual lesions may readily respond to treatment. There is no systemic disturbance throughout the attack, no fever nor loss of condition.

Positive diagnosis is arrived at by the demonstration of the sporothrix by cultural methods. The agglutination test, according to Widal, is also available.

Differential Diagnosis.—The principal conditions from which sporotrichosis must be distinguished are cutaneous glanders and epizoötic lymphangitis. From glanders it can be distinguished readily by its negative reaction to mallein, and from epizoötic lymphangitis by the absence of the cryptococcus on microscopic examination. Moreover, in sporotrichosis the lymphatic glands are not involved and there is no distension of lymphatic vessels (farcy-pipes).

Treatment.—Since the disease is infectious for man, horses, rats and mice, suspects should be at once isolated and steps taken to disinfect anything which may have been contaminated from the lesions, such as harness, grooming utensils and clothing, soiled litter, etc. Attention should also be paid to the extermination of rats and mice as possible carriers.

Curative measures consist of the internal administration of potassium iodide in full medicinal doses, and the local application of tincture of iodine with or without the previous light application of the actual cautery to fresh lesions.

G. H. W.

ULCERATIVE CELLULITIS

Ulcerative Lymphangitis ; Ulcerous Lymphangitis

Ulcerative lymphangitis is a contagious disease of horses, asses, and mules, usually running a chronic course and characterized by inflammation of the subcutaneous lymphatic vessels with the formation of buds followed by ulceration. The affection is not rapidly contagious, but is most easily spread amongst animals affected with parasitic skin diseases or injuries, and may thus spread more rapidly in large studs where there is a constant interchange of horses, such as remount depôts.

Etiology.—The cause of this disease is a small bacillus identical with the Preisz-Nocard bacillus which is responsible for a variety of conditions such as contagious acne in horses, and a pseudo-tuberculosis of the lungs and caseous lymphadenitis of sheep. Natural infection takes place by the introduction of the organism through a wound or abrasion such as cracked heels, itchy legs, and traumatic injuries as kicks. Infection may be conveyed by grooming utensils, bandages, infected litter, and even harness. The spread of the disease is facilitated by dirty conditions and general neglect.

Symptoms.—The disease most commonly affects the hind limbs, usually commencing in the neighbourhood of the fetlock. The part may become somewhat swollen and œdematous, but only slightly painful. Small buds or abscesses develop, varying in size from a bean to a walnut; they soon burst and discharge a thick creamy pus. This may be followed subsequently by a yellowish viscid discharge, which may be occasionally tinged or streaked with blood. After bursting the lesion becomes converted into a somewhat flat, red, granulating ulcer, with prominent margins. The ulcers tend to heal spontaneously in from ten to twenty days, that is much quicker than the cutaneous lesions of glanders or epizoötic lymphangitis. As certain lesions heal, others are produced, usually a little higher up the limb, and in this way the disease may continue for months or even a year. Between the lesions and above them the superficial lymphatic vessels may become swollen and prominent, but this is not always the case. Lameness occurs when the lesions involve the immediate neighbourhood of a joint or a tendon sheath, while sometimes the abscess may actually penetrate a tendon sheath and cause a permanent thickening of the part. There is little or no tendency to invade the lymphatic glands of the part beyond the production of slight swelling, and there is no tendency for suppuration of the glands. Constitutional symptoms, however, occasionally occur with an elevation of temperature to about 104° F. As a rule the appetite is not impaired, and good bodily condition may be maintained except in malignant cases where the lesions are very numerous. In those cases it may cause emaciation and exhaustion and terminate fatally.

Lesions are sometimes met with in unusual situations, such as the perineum and inside the thighs, sometimes in front of the shoulder and in the neck. It is rare for internal lesions to be produced, but they have been observed on post-mortem examination in the kidneys and in the lungs.

Diagnosis.—Ulcerative lymphangitis must be distinguished from glanders and from epizoötic lymphangitis. The lesions themselves may afford a sufficient basis for positive diagnosis, particularly in the absence of any affection of the lymphatic glands of the part. Moreover, the lesions have a much greater tendency to heal than in either glanders or epizoötic lymphangitis. On microscopic examination diagnosis is established by the presence of the characteristic bacillus which will stain by Gram's method, thus differing from glanders, and by the absence of the cryptococcus of the epizoötic lymphangitis. In addition an affected animal fails to react to the mallein tests for glanders.

Prognosis.—Ulcerative lymphangitis is not a very fatal condition, and when it does prove fatal death is probably due to secondary lesions in the kidneys or lungs. The course of the affection is irregular and somewhat treacherous in that recovery may appear to be imminent or even to have been effected only for a recurrence to take place and the patient soon to be as badly affected as ever.

Treatment.—In many cases a cure can be effected by lightly curetting the lesions and subsequently treating them with ordinary disinfectant lotions such as iodine solution, eusol, corrosive sublimate, or permanganate of potash. On the other hand, many cases so treated recur or fresh lesions develop and the case may become chronic.

Far better results are obtained by the use of vaccines prepared from the Preisz-Nocard bacillus of equine origin. If taken in hand early this method of treatment will usually result in a cure within a month (Watson). In this way not only is the troublesome affection removed, but unfavourable sequels such as permanent thickening of tendons, ankylosis, and elephantiasis are less likely to be developed. The vaccine is prepared by killing the organisms with alcohol and ether, and is injected subcutaneously under the skin of the neck and repeated every seven days in gradually increasing doses until five or six injections have been made. The injection produces a slight local swelling the next day, which soon disappears, and is less marked after subsequent injections. While the treatment is progressing the lesions dry up and the suppuration decreases, while the limbs become less swollen and more supple. The vaccine method of treatment should not be resorted to in cases of great debility, or if it is it should commence with very small doses, and only gradually increasing. Exercise should be given throughout the course of treatment, and the lesions should be kept clean, protected against flies and secondary infections.

G. H. W.

SPORADIC LYMPHANGITIS IN HORSES

Big-leg ; Weed ; Monday-morning Disease ; Gout ; Inflammatory Œdema

This is an affection peculiar to horses characterized by an acute fever and inflammation of the lymphatics of one or more limbs. Hind-legs are more frequently affected than fore-legs. It may affect any breed of horses, but is most frequently met with in heavy draught horses, and particularly those with puffy limbs, generally known as gummy-legged horses.

Etiology.—The common cause of the condition is infection with micro-organisms which have gained access usually through small wounds on the limbs as from kicks, brushing, cracked heels, grease, and also bad thrushes. In many cases, however, it is impossible to discover any wound from which infection may have occurred. Such cases remain obscure, and it has been suggested that the seat of infection may have been the alimentary tract and the abdominal lymphatic glands. Such an explanation, however, fails to account for those cases occurring under similar conditions in the fore-limbs.

There are certain predisposing causes which are frequently in operation in cases of Weed. The most frequent one is rest from work and exercise for one or two days, during which the horse has received his usual ration, hence the frequency with which it occurs in studs of working horses after a week-end rest, giving rise to the name Monday-morning Disease. At other times it is associated with a change from a comparatively low diet to an excessively high nitrogenous diet, such as by the inclusion of too large a proportion of beans or peas, etc. It is probable that these factors bring about a slowing down or partial stasis of the lymphatic circulation, particularly of the extremities, resulting from depression of the heart and general circulation. This causes puffiness of the limbs and allows the organisms, which may be present in a small wound, to assume greater activity and penetrate into the lymphatic vessels and so set up inflammation of them. Dark, dirty stables which favour the existence of dirty legs may also be regarded as one of the predisposing causes, though the affection is sometimes met with in stables with good hygienic conditions.

Symptoms. — The symptoms are usually ushered in with rigors or shivering fits, and fever frequently up to 105° or 106° F. at the outset. In recurring cases, however, the temperature is rarely so high. The respirations are accelerated, the mouth hot, the mucous membranes injected ; pulse is a typical febrile pulse and is full and bounding. Acute lameness then develops, often affecting one limb and that most frequently a hind-limb, occasionally a fore-limb, which is swollen, painful, and very hot to the touch, and often held up off the ground. Beads of yellowish glairy liquid may be seen on the skin of the affected leg. The swelling usually commences at the top of the limb, and the inguinal lymphatic glands inside the thigh or the axillary lymphatic glands inside the armpit are found to be swollen and hot, and very painful to manipulation. The acute swelling in this region causes an abduction in the limb, and the patient can only with difficulty be made to move. The swelling then extends down the inside and gradually around until the whole limb down to the foot is involved. In some cases the swollen parts will pit on pressure. As the swelling increases the pain frequently subsides. In the painful stage the horse may break out into patchy perspiration, refusing all food but is usually very thirsty. Constipation is almost always present, fæces being firm and mucus-coated, and the urine is diminished in quantity but is markedly raised in specific gravity.

An animal affected with acute lymphangitis usually stands throughout the course, but in cases where the pain is excessive he may lie down and then has considerable difficulty in rising and bed-sores may develop. In a few cases suppuration occurs as a complication. Abscesses may form in the lymphatic glands or along the course of the lymphatic vessels on the inner side of the limb. Œdema may also extend under the abdomen, involving the sheath in males and the udder in mares.

The acute stage of the condition may last one or two days, and in favourable cases the pain and swelling gradually subside in the course of a week or ten days. In some cases it may persist for several weeks.

The condition is often recurrent and horses that have had one attack appear to be very susceptible to second attacks. Recurrences generally lead to a permanent thickening of the subcutaneous connective tissue surrounding the lymphatics, producing a chronic thick leg and giving rise to the term “ elephantiasis.” It should be noted that the swelling in the acute stages is due to the accumulation of lymph in the lymphatic vessels and connective tissues, while chronic thickening is due to increased connective tissue. The lymph never becomes organized, that is, converted into cellular tissue, but it may become replaced by connective tissue.

Treatment. — *Prevention.* — In the cases of animals compelled to remain in the stable a day or two, the diet should be reduced, the corn being substituted with a bran mash to which may be added a little alterative or diuretic such as potassium nitrate and sulphur sublimate āā ʒiii. and black antimony ʒi. It is a good plan to adopt this procedure as a routine measure at week-ends. In the case of animals subject to the condition care must be taken with the diet in general, seeing that it is never too highly nitrogenous. The bowels in such cases should be kept in a semi-relaxed condition, and on days when the animal is not at work he should be taken out for a little walking exercise. Brushing wounds, cracked heels, and other abrasions should be consistently disinfected.

Curative Measures.—If seen in the earliest stages bleeding may be practised, but this is not so frequently done now as formerly, and in the opinion of the writer equally good results may be obtained without resorting to that operation.

One should begin with the administration of a full dose of physic, giving aloes ʒiv.-viii. according to the size of the horse and the climatic conditions. The large dose is usually required in cold climates, the smaller in the hotter zones. This should be immediately followed with a combination of stimulants, febrifuges, and diuretics. A useful bolus consists of ammonium carbonate and potassium nitrate āā ʒii. and pulv. digitalis ʒss. Potassium iodide may also be given with advantage as it is an internal antiseptic and tends to diminish the thirst and reduce the swellings. In very painful cases an analgesic may be given, such as tincture of aconite and salicylates. Locally hot fomentations should be applied by means of a large piece of flannel or blanket dipped into very hot water and wrung out almost dry. The latter point is essential, otherwise the skin may be injured. The leg should then be massaged in the upward direction and a straw bandage put on the limb, commencing at the foot and extending as high as possible up the limb. If properly applied this affords very considerable relief. Sedative applications are sometimes preferred to the fomentation, such as lotions of lead and opium or lead and belladonna, to which a little glycerine is added to assist penetration. The horse should be placed in a loose box with short litter so that he can take voluntary exercise if he wishes, and as soon as he is able to move the limb he should be given light walking exercise for ten or fifteen minutes several times daily. A search should be made for any wounds, which must be thoroughly disinfected, and any abscess that may have developed should be evacuated and similarly treated. The diet should be somewhat restricted and laxative, such as linseed mashes, scalded oats, and a little good hay and green meat if available.

In chronic cases it is difficult or impossible to produce any reduction in the thickening of the limb. A course of potassium iodide ʒii. once or twice a day, or biniodide of mercury grs. v.-viii. dissolved with the assistance of grs. xv. of potassium iodide once daily, have frequently been recommended, but they are of doubtful benefit. The hypodermic injection of fibrolysin may be tried and is sometimes effective in causing some reduction in the size of the limb if the thickening is not of too long standing.

Animals affected with chronic big-leg should be kept constantly at reasonable work, otherwise there is a great tendency for recurrence, and if kept indoors the limb invariably swells more. Obviously no horse should be worked, however, in the acute stages of lymphangitis or during a relapse.

G. H. W.

BURSATI (BURSATTEE)

Bursati is the name used in India to designate a particular diseased condition of the skin and subcutaneous tissues of equines which is characterized by the formation of fibrous tumours embedded in which are peculiar bodies known as *kunkurs,* and ulceration of the overlying skin.

The occurrence of conditions more or less closely resembling bursati in their clinical features has been reported in countries other than India. Some of these have been considered as identical with bursati, but proof of this has not yet been furnished. The existence of these conditions and their identification with bursati has led to a considerable amount of divergence of opinion as to what are the clinical features of the disease.

The first reference to bursati in veterinary literature appear to be those by Kerr (1829) and Tombs (1831). Both these authors attributed the disease to poverty of condition and to atmospheric influences. In 1842 the view was put forward by Jackson that the disease is due to a fungus. In 1853 it was suggested by Hodgson that the condition is of a cancerous nature. Between the years 1872 and 1885 several papers were published (Spooner Hart 1872, Collins 1874, Cane 1876, Meyrick 1878, F. Smith 1874 and 1885, Burke 1880 and 1884, and Steel 1885) in which the disease was attributed to one or other of the above causes. In Meyrick's paper the suggestion was put forward that flies played some part in the transmission of the disease; a view that has long been held by the natives of India.

In 1905 Lingard published a monograph on the filarial embryos found in the blood of horses and cattle in India and stated that bursati was caused by them. In his view the lesions resulted from the impaction of embryos in the capillaries, and he claimed to have found immense numbers of these parasites in them. He further stated that the lesions were not confined to the skin, but occurred also in the internal organs. These views were supported by Place in the following year.

The principal *clinical characters* of bursati are as follows :

The disease has a seasonal distribution, the immense majority of the cases occurring during the rainy season, June to September or October, as is indicated by the name, which is derived from " bursat," meaning " rain."

Bursati lesions tend to heal with the onset of the cold weather, but may reappear the following year. The parts of the body most frequently involved are the pasterns and fetlocks, neck, withers and shoulders, lips, eyelids, penis and prepuce, and vulva.

The first evidence of disease is the development of a hot painful swelling in the subcutaneous or submucous tissue, the overlying skin or mucous membrane remaining uninvolved. Within about a week the swelling becomes much harder, and shortly after this the skin becomes thickened and adherent to the underlying tissues. A fortnight or three weeks later the skin covering the lesions sloughs, leaving an open sore, the margins of which are thickened and fairly regular. Yellowish red granulations develop over the ulcerated surface which becomes raised above the level of the surrounding skin. This elevation is mainly due to the development of fibrous tissue in the subcutaneous tissue. There is practically no pus formation in uncontaminated lesions, only a very small amount of thin yellowish liquid collecting on the surface. Embedded in the ulcerated surface are usually found a number of small rounded or irregular bodies, which can be enucleated by pressure, leaving small cavities. These bodies, which are known as " kunkurs," vary in size from a pin's head to a pea. In the earlier stages of the disease they are of a dense fibrous consistency, but later may undergo partial calcification. Lesions that are not exposed to friction or injury show little or no tendency to spread and are generally covered with a scab, but lesions that are situated in places exposed to irritation of any kind often attain a very large size. Cases are on record in which the fibrous masses comprising bursati lesions of the pastern or fetlock have become as large as coco-nuts.

As already stated, flies have long been considered by the natives of India to play a part in the transmission of bursati, and this view is to some extent supported by the fact that bursati lesions frequently develop in connection with wounds and in situations that are exposed to injury. Holmes stated, however, that in his opinion this is not the most common origin of bursati lesions for the reason that the lesion is primarily one of the subcutaneous tissue and not of the skin, involvement of the skin being secondary. Holmes was also of the opinion that while debility and atmospheric conditions are not the direct causes of the disease, they do play a secondary part in its production.

The lesions, whether small or large, are composed almost exclusively of dense fibrous tissue which merges gradually with the subjacent tissues. If a lesion be divided there are found embedded in it kunkur bodies similar to those not infrequently found embedded in the surface under the scab. These are more or less irregular in shape, and, in more recent lesions, of a dense fibrous consistency. When bursati lesions occur in the submucous tissues they appear to be confined to the visible mucous membranes.

A number of authors have described the occurrence of bursati lesions in internal organs such as the liver, but Holmes was never able to satisfy himself that these were true bursati lesions, and is inclined to think that mistakes in diagnosis have been made, such lesions being due to the presence of worm parasites.

Microscopic examination of sections cut from bursati " tumours " shows that they are composed almost exclusively of fibrous tissue, and consequently the histological details vary somewhat with the age of the formation. In the earlier stages the stroma is more or less loose in texture and has enmeshed in it a large number of cellular elements—fibroblasts, polynuclear and mononuclear leucocytes. There is also a marked infiltration with eosinophile leucocytes. In the older lesions the matrix is more dense and the cells are less numerous. Giant cells are not infrequently found, and the eosinophile cells are frequently massed together in groups. A few cells containing pigment may be found. There is never any evidence of caseation. Embedded in this fibrous matrix are the so-called kunkurs. It is often difficult to keep these in place in sections as they are very liable to drop out during the processes of staining. The kunkurs appear to be surrounded by a thin fibrous capsule special to them. In the early stages the kunkurs are composed almost entirely of cells the great majority of which are eosinophile leucocytes. These are included within the meshes of a delicate fibrous matrix. In the older lesions the kunkurs more closely resemble the general mass of the " tumour " in minute structure but may show slight calcification.

Etiology.—The principal views that have been put forward regarding the nature of the specific cause of bursati are (1) that it is due to worm embryos, (2) that it is caused by a fungus. The first of these views was expressed by Lingard and supported on insufficient grounds by Place. The second has been supported by a number of authors.

In spite of what has been written by these authors regarding the causation of bursati, it cannot be said that actual proof that the disease is caused by a fungus has been furnished. Holmes was able to detect a fungus in specimens that had been macerated in 40 per cent caustic potash. Pieces of the " tumour " were macerated for sixteen to twenty-four hours and fragments of this were pressed between two slides. In such preparations isolated spore-like bodies and broken strands resembling mycelia could be found, but it is said that it was not possible to differentiate these satisfactorily from fragments of nuclei, granules of eosinophile cells, and tissue fibres. It is further stated that in preparations from young lesions very convincing pictures of the spores and fibres could be found. Success was not obtained when attempts were made to demonstrate these forms in stained specimens.

Attempts were made to obtain cultures from bursati lesions and also from the blood of diseased horses. The most constant result obtained was the development of a slow-growing fungus on Sabouraud's medium. Specimens of this were examined by Butler, who found that the organism resembled *Sporotrichum minutissimum*, which is believed to be the cause of a skin disease in man. Horses, rabbits, and guinea-pigs were inoculated subcutaneously and intravenously with emulsions of natural lesions and with cultures. In every case the result was entirely negative.

In his conclusions Holmes states that his investigations afford some evidence that bursati is a mycosis somewhat resembling Sporotrichosis of the horse and mule described by Carougeau in Madagascar, but does not claim to have established the fungus found by him as the cause of the disease. From the illustrations given in Holmes's paper it would appear that the sporothrix responsible for the disease in horses and mules is a very different organism, and a further distinguishing point is the ease with which cultures of the sporothrix responsible for lesions in equines can be obtained on agar and potato and in broth at ordinary room temperature.

Treatment.—It is advised that the fibrous tumours should be excised, the wound being treated by ordinary methods. The actual cautery or the application of caustic dressings does not yield good results. Holmes's experience was that not all cases yielded to the same treatment. In some cases good results followed the administration of arsenic, either alone or in combination with atoxyl. Three courses of ten doses were given, an interval of one week elapsing between the courses. Holmes does not consider that arsenic is in any way a specific, his view being that the good results are due to the general tonic effect of the drug.

A similar method of treatment, in which doses of biniodide of mercury ranging from 5 to 20 grains with 1 to 3 drachms of potassium iodide were substituted for the arsenical salts, also gave good results in some cases.

Hazelton states that after complete surgical extirpation of all fibrous tissue the wound should be kept constantly dressed with red oxide of mercury; liquid dressings are to be avoided. He also recommends that cases that show little tendency to improve should be removed to another station.

Although there is no evidence to indicate that the disease is transmitted from animal to animal by any outside agency, it is advisable to isolate affected animals.

Diseased Conditions that resemble or have been identified with Bursati

Leeches or Leeching.—This condition occurs in the United States of America. Moore, in his text-book, *The Pathology of Infectious Diseases of Animals*, definitely gives " bursati " as a synonym for " leeches." As the name indicates, the disease occurring in America was thought to be due to or in some way connected with the water-leech. Neal (1885–1887) states that the disease makes its appearance on parts of the skin which have been submerged in ponds, although he adds that the lesions are sometimes met with on the head, *e.g.* involving the eyes or the mouth. The lesions bear a close resemblance to those of bursati as seen in India. In 1886 Lyford described leeches as occurring in horses and mules, and connected the disease with marshy ground and the rainy season.

Bilting, writing in 1894, stated that the term " leeches " covered two distinct conditions, one due to the liver-fluke and the other closely resembling bursati, the word " leech " referring to the bodies known as kunkurs in India. He considered the lesion to be a round-celled sarcoma.

In 1897 two papers on " leeches " appeared (Fish and Smith). Both of these authors attributed the disease to a fungus. The former author drew attention to the similarity existing between leeches and bursati.

In the same year Thomson wrote as follows: " The ' leeches ' are of two forms. One has the shape of a grain of whole rice with the hardness of stone. The hard grit causes a knife to become dull. They grow sometimes in clusters as long as a small finger. The other forms patches of a gristly nature from the size of a large pin-head up to the size of a finger-nail, flat and of irregular shape."

The condition described by Griffin as "leeches" or "hoof-rot" occurring among mules in Florida appears from the clinical description to be an entirely different disease. This was considered to be due to the penetration of the skin around the coronets by a "water insect" that was said to resemble a screw-worm or small bot.

Summer Sores, Granular Sores, or **Granular Dermatitis.**—This condition is identified by Law, Moore, and Fleming with bursati, a view which is upheld by Lingard.

It is possible that the various authors who have written regarding "summer sores" have not all been studying the same condition.

"Summer sores" were first described by Bouley in 1850 and in 1868. Rivolta claimed to have established *Filaria irritans* as the causal agent. Querrua and Laulanie confirm this.

In view of the now apparently established fact that worm parasites are not causally connected with bursati in India, it would appear that "summer sores" is not identical with that disease.

Swamp Cancer.—This disease is described by Gilruth as occurring in the Northern Territory of Australia, and it is also known to occur in Queensland and North-West Australia. This author draws attention to the resemblance that this disease bears to bursati in India and to leeches in the United States.

A. L. S.

BOTRIOMYCOSIS

Discomycosis

Botriomycosis is a specific disease affecting horses and characterized by the formation of a tumour-like lesion called a "botriomycoma," which consists largely of a chronic inflammatory proliferation of connective tissue with suppurating foci.

Etiology.—Botriomycosis is caused by a micrococcus called *Botriomyces ascoformans,* which in the lesions develops in colonies known as botriomyces or discomyces granules. These colonies consist of a number of micrococci closely packed together. Individually and culturally they are indistinguishable from *Staphylococcus pyogenes aureus.*

The disease is comparatively common, and is the result of a wound infection. The wound may be an apparently insignificant one, as a scratch by some part of the harness such as the collar, or a more serious operation wound, as in castration. The affection may also occur in various parts of the skin and in the udder of mares. Generalization is rare, but has been recorded with secondary lesions in the lungs, spleen, liver, kidneys, and lymphatic glands. It is a sporadic affection occurring generally in isolated cases and showing little or no tendency to spread to other animals in the stable. It is so slow of development that horses castrated as yearlings and inoculated then frequently do not show symptoms of the disease until they are five or six years old or even older. It is very rarely a fatal disease, except in those few cases of generalization, but horses affected may be rendered unfit for work, either by interference with some part of the harness, or when the spermatic cord is the seat of the lesion by interference with the gait and often producing lameness.

Symptoms.—The symptoms are almost entirely limited to the lesions. The latter consist of tumours which may be single or multiple. Their size varies considerably. They may be the size of a pea or as large as one's head or even larger. Their common sites are in the front of the shoulder (shoulder tumour), spermatic cord (scirrhous cord), the point of the elbow (infected elbow tumour or shoe-ball), the tail, the skin, and the udder, while the same causal organism has also been found in lesions of poll-evil.

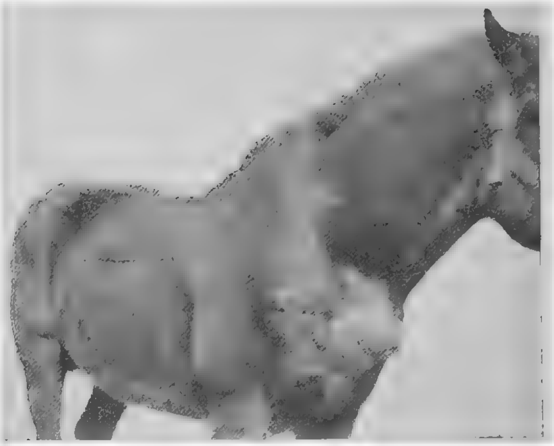

FIG. 9.—Shoulder tumour in horse due to botriomycosis.

The name "mycofibroma," which is sometimes applied, fairly well indicates the common characters of the lesions. The main substance of the new growth is dense fibrous connective tissue, firm on manipulation, containing a varying number of suppurating foci, which are virtually abscesses. The skin over the enlargement may point and fluctuate and then burst, emitting a sticky kind of pus. The wound thus produced may then heal, but fresh abscesses develop and repeat the process. Sometimes there are several openings all communicating with purulent centres.

When the *shoulder* is affected (shoulder tumour) the lesion may be purely subcutaneous or it may be deep-seated and extend into the levator humeri muscle, and sometimes completely surrounds the jugular vein. In some cases the seat of the lesion is the prescapular lymphatic gland.

When the *spermatic cord* is the seat of the lesion (scirrhous cord) the new growth may be right at the end of the cord and completely in the scrotal sac, while in some cases it extends up the cord into the inguinal canal. In these latter instances one rarely realizes the extent of the condition and the difficulty of the surgical removal of the tumour until the patient is cast and a very thorough examination made. The tumours are well supplied with vessels and are generally closely adherent by fibrous tissue to the walls of the scrotum. It is quite common for several fistulæ to run into the centre of them.

The *stump of the tail* may also become affected and develop a characteristic frequently discharging tumour, which has been graphically described as "turnip tail."

The *skin* and subcutaneous connective tissue in various parts of the body may become affected with botriomycosis, such as the upper border of the neck in front of the withers, the quarters, and the buttocks. In such cases the lesions are numerous, about the size of hazel nuts, and extremely painful, and many of them discharge the characteristic pus in which the organism can be demonstrated microscopically.

The *udder* of the mare is sometimes involved, resulting in the formation of hard, dense lumps, or a nodular induration and with the skin closely adherent. In such cases the growth may extend to the inside of the thighs. Sooner or later discharging centres are produced as elsewhere.

The pus from a botriomycoma is characteristic in its appearance to the naked eye. It is sticky and tenacious, and if a little is spread on a slide a number of granules can be seen. Unlike the pus from actinomycosis, however, the granules do not give a gritty feel when rubbed between the thumb and fingers. They consist of colonies of the botriomyces or discomyces.

When any of the growths are cut into after removal they are found to be very dense and firm, almost tendinous in places both as regards their hardness and their glistening appearance. In other parts they look quite watery, and often show areas of yellowish fat. One or more cavities and channels of varying size may be seen, some very minute and others fairly large, and each containing the characteristic pus with granules.

Differential Diagnosis.—Botriomycosis must be distinguished from actinomycosis, which on rare occasions affects similar situations. The distinction is usually made by microscopic examination of pus from the lesions, revealing the characteristic colonies of cocci in botriomycosis and of clubs in actinomycosis. Scirrhous cords may also be encountered which are not due to botriomycosis. Their true character is, however, only discoverable by microscopic examination. They rarely discharge unless due to botriomycosis or actinomycosis.

Treatment.—Whenever a botriomycoma is sufficiently well circumscribed to make excision possible, it should be operated upon (see surgical section). The internal administration of potassium iodide has been recommended in many quarters, but it is by no means reliable. In the surgical removal of botriomycomata there is usually considerable hæmorrhage, and it is advisable to practise ligation freely. The entire growth should be removed wherever possible, otherwise it may recur. Occasionally, however, partial excision yields satisfactory results. If the growth is inoperable tincture of iodine may be injected into the substance of the tumour and a smart blister of biniodide of mercury applied externally. This may cause a reduction in the size of the growth to operable proportions. The injection of fibrolysin may also bring about a reduction in size of the growth. In more recent cases puncture of the enlargements, curetting and packing with wool soaked in tincture of iodine will sometimes bring about disappearance of the tumour.

In the diffuse superficial form with multiple suppurating points treatment is very difficult. Good results are sometimes obtained by inserting into the centre of each suppurating point for several successive days a grooved director which has been previously dipped in pure tincture of iodine or pure carbolic acid. If such lesions, however, are very extensive treatment is rarely justified by results.

With regard to botriomycoma of the udder, Fröhner says that in his experience it is a very malignant tumour, and that operative removal is liable to be followed by recurrence. For this reason he recommends total amputation of the udder, even where the disease is confined to one half of it. In cases seen by the present writer the skin and subcutaneous tissues of the groin and inside the thigh have been so involved as to render any form of treatment valueless.

G. H. W.

TETANUS

Lockjaw

Tetanus is a specific disease caused by the *Bacillus tetani*, which is introduced into the body by way of a wound. All classes of animals are susceptible, but horses are much more frequently affected than other species. The organism exists more in some localities than others, but has a very wide distribution. Its natural habitat is the soil, and it is probably commoner as a soil organism in tropical than in temperate or cold climates. It is also frequently present in the fæces of herbivora.

Etiology.—The organism of tetanus is an anaerobic bacillus, rod-shaped, and forming spores which distend the end of the bacillus, giving it the appearance of a drum-stick. It is a tissue parasite, and remains at the seat of inoculation, where it produces a toxin which is readily absorbed mainly by the nervous system, its effects varying according to the particular part of the nervous system affected. It appears to produce no local inflammation, although it is frequently accompanied by other organisms which probably favour the action of the tetanus bacilli. As already stated the organism is probably always introduced through a wound. Where the wound is discoverable it is said to be **traumatic tetanus,** but where the wound is indiscoverable it is sometimes termed **idiopathic tetanus.** Owing to the common habitat of the organism, wounds of the limbs are more liable to infection than those of other parts of the body. It thus most frequently follows cracked heels, punctured wounds of the foot, picked-up nail, and also castration and docking. Infection may also occur at the navel in the newly born. Some idiopathic cases may possibly be of intestinal origin through injuries inflicted by worms, while at other times it may follow parturition through injuries to the uterine or vaginal wall.

The *period of incubation* of tetanus is from one to five days, although it is sometimes as long as ten or fourteen days, or even twenty days, while the course varies from three to fifteen days after the onset of symptoms. It frequently happens that recovery occurs if the patient can be kept alive for ten days, but the period of convalescence may extend over several weeks. Recovery from one attack confers an immunity which lasts over a year.

Symptoms. — Symptoms usually commence with a stiffness affecting various parts of the body. The animal moves his limbs with difficulty, and horses are inclined to drag the straw about with their feet. Feeding is slow and may later become impossible. The respiration is accelerated, the pulse becomes small and hard. The temperature varies, in the majority of cases being from 103° to 104½° F., but sometimes as high as 108° F. The patient is very excitable, and often sweating in patches. As the condition advances the fore-limbs are usually drawn widely apart, and the hind limbs may be in a similar position or drawn forwards under the body. The patient is unable to turn round in the box, or does so with very great difficulty. Various sets of voluntary muscles become affected with spasm, the most common being those about the head. The nose is outstretched and the nostrils expanded, and the lips become drawn and teeth exposed, while the ears are often drawn together at their tips; the membrana nictitans or "haw" protrudes over the eye, which is often sunken and fixed. This protrusion is more easily observed when the patient is tapped under the chin and made to raise his head quickly. When the muscles of the jaw become involved it is impossible to open the mouth and mastication ceases, swallowing is difficult, and dribbling is set up. This closure of the jaw is called **trismus.** In those cases where trismus does not occur some appetite usually remains. In other cases the hind, quarters are affected before the head, the tail being raised and quivering. When the muscles of the back are affected, raising the head and tail, the condition is called **opisthotonos.** When the lower part of the body is affected, drawing the head and tail downwards and arching the back, it is called **emprosthotonos.** If one side of the body is affected, producing a lateral curvature, it is termed **pleurothotonos.** During the course of tetanus the bowels are more or less inactive. Tetanic patients are very sensitive to light and sound, and if disturbed are frequently thrown into increased spasms, during which they may fall to the ground. Tetanic patients rarely lie down, and if horses do go down the case is almost invariably fatal.

Pneumonia often sets in as a complication, and this is generally due to saliva, etc., running down the trachea owing to the difficulty in swallowing.

In **cattle** the general symptoms are very similar to those of horses, and trismus usually exists. Protrusion of the membrana nictitans is marked as in the horse. There is the general muscular stiffness and not infrequently tympany.

In **sheep and goats** similar symptoms are noticed, though these animals usually lie and rapidly become tympanitic. In tetanus in young lambs infection is fairly common by way of the navel, and also after docking, and a number may be affected simultaneously. The characteristic muscular spasms are present and the disease is almost always fatal.

In **pigs** the head and neck are most commonly affected, with frothing at the mouth and general symptoms as in other species.

In **dogs** tetanus is not very common, and yet not very infrequent. The limbs are usually outstretched and the head erect, the eyes are frequently drawn to one side or the other (squinting). The closure of the jaws is varied —in some cases very pronounced, at other times only indicated by some stiffness. If the patient is touched or approached severe spasms are set up.

The *mortality* of tetanus is very high. In sheep and swine nearly all cases prove fatal. In horses and cattle from 20 to 25 per cent recover. Death is usually due to either pneumonia, asphyxia, or exhaustion.

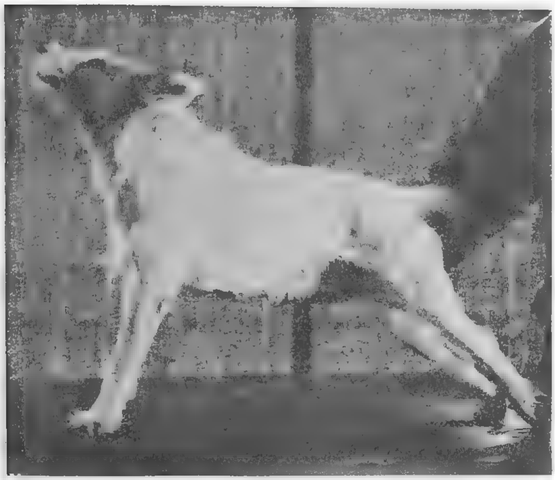

Fig. 10.—Dog with tetanus.

Differential Diagnosis.—The following conditions must be distinguished from tetanus:

Strychnine Poisoning.—In this affection the spasms are clonic and the course is much more rapid.

Acute Laminitis and Acute Rheumatism.—In each of these affections there is great stiffness and indisposition to move about. In the former there is an absence, however, of muscular spasms, the feet are hot, and the digital arteries throbbing, and in the latter condition the muscles are painful and tense. In each case there is an absence of trismus, and the membrana nictitans does not pass over the eye.

Acute Nephritis.—In this condition the patient often stands in an attitude resembling tetanus and often refuses to move, but there is the absence of muscular spasms, and the haw does not pass over the eye.

In **Cerebro-spinal meningitis** there may be rigidity of the neck, but there is no trismus, and there is a general dulness and depression. Moreover, tonic muscular spasms are not brought on when the patient is excited as in tetanus, although hyperæsthesia is frequently present.

In **Tetany** groups of muscles either of the neck or limbs, or occasionally the jaw, become affected with tonic spasms. The condition, however, usually passes away fairly quickly, but may be recurrent.

Treatment.—Prophylaxis.—In districts where the disease is known to be prevalent prevention should be practised, particularly in the case of valuable animals. All wounds should be treated antiseptically and a good drainage should be provided. Anti-tetanic serum should also be injected. This should be done before all operations in such districts, and as soon as possible after accidental wounds. The dose of the serum should be about 500 American units for a horse or ox, which is the amount usually present in c.c. x. of standardized serum. This produces a passive immunity for about three weeks. To be on the safe side, therefore, the injection should be repeated in a fortnight. In the case of accidental wounds the sooner the serum is injected the greater is the likelihood of successful prophylaxis.

Curative Measures.—The same principles must be adopted in any species affected. In the first place the seat of inoculation should be sought and thoroughly disinfected. This may necessitate excision of the tissues round the wound, or where that is impracticable the application of a caustic such as silver nitrate or perchloride of mercury. If the infected wound is the tail after docking, the animal should be docked again a little higher, and the wound seared with the actual cautery. The patient should then be placed in a dark, quiet box, and should be disturbed by nobody except the usual attendant, who should also proceed very quietly. In the case of horses slings may be used, but only in animals that are not too excitable, otherwise the symptoms may become aggravated.

Attention to food is important, and so long as the patient is able to eat he should be given soft, concentrated, highly nitrogenous foods, such as boiled oats, linseed and bran mashes, and a little green food, and water should be within reach. If the animal is unable to masticate, thin gruel or milk should be left within reach so that he may take small quantities when so disposed. At the same time peptonated milk and eggs may be administered as an enema, and in the case of small animals nutritive suppositories may be given.

Medicinally, the tendency to constipation should be controlled by means of a laxative, such as sulphate of magnesia in the drinking-water, or the preliminary administration of calomel, which may be given in the food or in the form of electuary. It is distinctly unwise to administer a bolus, as the excitement may suddenly produce trismus.

There is no curative agent as satisfactory as anti-tetanic serum as a preventive. The serum is apparently of very little service when once the symptoms have been established. Its action is to neutralize the toxin, but when the toxin has already exerted its influence on the nervous tissues the serum is of no avail. It is possible, however, that repeated injections of serum may neutralize some of the freshly produced toxin, and so check the progress of the symptoms. Moreover, it is probable that much larger doses should be given to produce any appreciable curable effects, probably from fifteen to twenty times the effective preventive dose. This amount is rarely available, and the expense entailed would probably be objected to by the owner, particularly if the termination of the case were unfavourable.

Of drugs the most serviceable are those which reduce muscular spasms, and probably the best of these are belladonna and cannabis indica in full medicinal doses, repeated two or three times daily. Chloral hydrate and potassium bromide are also useful to reduce excitability and spasms. Other agents that have been recommended include perchloride of iron, hydrocyanic acid (ʒi.-ii. for horse or cow in a little water per rectum). Carbolic acid injected subcutaneously and iodine solution similarly administered have been strongly recommended, but their efficacy is not well established. A more promising line of treatment is the subcutaneous injection of sulphate of magnesia. It has already been shown that intraspinal injections of this agent are capable of controlling the convulsions and spasms of experimental tetanus in monkeys, the effects lasting for twenty-four hours or longer. This method of administration is scarcely practicable in the larger animals, and hypodermic injection has therefore been carried out in a number of cases with promising results. The dose administered is c.c. xl. (about $1\frac{1}{2}$ ounces) of a saturated solution injected half on each side of the neck. This is repeated twice daily for ten days and once daily for another ten days. Some relaxation of the muscles may be observed within two hours of the injection. There is no marked general improvement in the condition of the animal, however, until the fourth or fifth day, when the symptoms become less severe and the trismus less pronounced, excitability is reduced, and the patient is better able to take a little food. These results have been obtained in a few cases in both England and America, and although they are highly encouraging, they are not yet sufficiently numerous to justify general conclusions.

The period of convalescence after recovery from tetanus should extend over several weeks. During the earlier part of the time the patient may be given a little walking exercise for fifteen to thirty minutes, which may be gradually increased; and after the horse appears to be completely recovered, a full conditioning course should be carried out.

G. H. W.

MALIGNANT ŒDEMA

Traumatic Gangrene ; Gas Gangrene

Malignant œdema is an acute bacterial affection due to the inoculation of wounds with a bacillus—bacillus of malignant œdema (Koch), vibrion septique (Pasteur). (Recent researches, however, go to show that the disease known by the name of gas gangrene in the human subject may be set up by a series of anaerobic organisms, of which the above may be one, acting independently or in combination.) It may be met with in all domesticated animals, the most susceptible being the horse. The ox, however, has a very great degree of resistance, and by many authorities is said to be immune.

Etiology.—The bacillus of malignant œdema has a world-wide distribution, and is more or less constant in soil. It is a rod-shaped anaerobic organism, somewhat resembling the anthrax bacillus, but much more slender. In the body several bacilli may be attached end to end and may sporulate, the spores being oval in shape in the centre of the bacillus which it distends. It stains readily by aniline dyes, but not by Gram's method. As a cause of disease the bacillus in malignant œdema is met with only in the tissues. It is, however, more or less constantly present in the alimentary tract, and very rapidly invades the blood-stream after death, in which situation it has to be carefully distinguished from the bacillus of anthrax. The natural method of infection is through a wound of the skin or mucous membrane, and infection may occur as the result of contamination with dirt, soil, putrid matter, and excreta. It rarely sets up disease unless introduced into structures deeper than the skin, and consequently is a common sequel to lacerated wounds. It may also be introduced by hypodermic injection as the result of dirty procedure, unsterilized syringes, etc. In the horse it is not an infrequent complication of castration wounds as the result of which it may set up septic peritonitis, which may similarly occur after parturition as the result of injuries to the uterine wall.

Symptoms.—After the infection of a wound the tissues become swollen, hot, and painful, and may crepitate on manipulation. The tissues become badly discoloured and assume a livid lustre, while a reddish-brown serous fluid, often frothy and fœtid, exudes; the margins of the wound may become necrotic. The temperature is somewhat raised up to 104° or 106° F. in the acute stages of the affection, but becomes sub-normal before death in fatal cases. The pulse is small and weak, and sometimes almost imperceptible, while heart-beats may be tumultuous.

When malignant œdema follows castration the condition may occur almost at any time prior to the healing of the wound. The infection of the cord causes an extensive œdema with great swelling of the scrotum, extending forward under the abdomen. The affected area is extremely painful, the patient is off its food, respiration becomes blowing, temperature elevated, pulse weak and small. A fœtid discharge exudes from the wound. This is often followed by diarrhœa, and in most cases the condition proves fatal in from twelve to forty-eight hours after the onset of symptoms.

In cows an infection said to be due to malignant œdema sometimes follows injury to the womb during parturition, the symptoms occurring two to four days later. The external genital organs become hot and swollen, at first painful but later painless, and a dirty reddish-brown fœtid discharge escapes. The condition proves fatal in from one to three days.

Treatment. — Prevention consists in the thorough disinfection of all wounds and their protection against contamination with dirt or soil, etc. Immunization may also be carried out by several methods, but as the disease is not very common they are of small practical value. In the human subject a polyvalent serum has been found to give excellent results if injected immediately after the infliction of a dirty wound.

Curative measures are essentially surgical, and consist of free incisions into the affected part and the excision of obviously affected areas. Thorough disinfection should be practised, amongst the best agents being iodine, hydrogen peroxide, eusol, carbolic acid, and lysol. The injection of anti-serum if available will check the extension of the disease, and may be followed by recovery.

Medicinal treatment is not of great service, but cardiac and general stimulants are indicated, such as digitalis, ether, and caffeine, together with quinine and salicylates as febrifuges and internal antiseptics.

G. H. W.

BACTERIAL NECROSIS

Bacillary Necrosis ; Necro-bacillosis

Under this heading may be considered a number of affections occurring under a variety of conditions and in different species of animals, and caused by the bacillus of necrosis (*Bacillus necrophorus*). The organism has a very wide distribution in nature, being commonly present in soil and in the elementary tract of various animals, particularly herbivora and swine. Cases of the disease, though not infrequent, are usually isolated, while at times the disease appears to be spread by contagion.

Bacillary necrosis is always an inoculation disease, that is to say, the organism requires some injury to tissues for its entrance into the body. For skin necrosis comparatively simple breaches of the skin are sufficient, such as chafing of the harness, burns, punctured wounds, injuries to the foot as in treads, etc. Eruption of the teeth or wounds of the mucous membrane of the mouth afford opportunities for the entrance of the organism. Similarly, slight abrasions of the mucous membrane of the alimentary tract may result in invasion by the organism. The bacillus is essentially a tissue parasite, but it is sometimes carried by the blood stream from the primary lesion, setting up secondary or metastatic lesions in other parts of the body.

The first effect of the entrance of the organism into the tissues is the destruction of any of the cells or tissues with which the toxins of the bacillus come into contact. The necrosis or death of the tissues spreads from this point fairly rapidly, and results in the formation of a compact mass more or less adherent to the surrounding tissues. From this point deeper tissues may become invaded, forming ulcers or fistulous tracts of a varying depth.

The local lesion, primary or secondary, may be described as a circumscribed patch of dull brown tissue, occasionally greenish white as when affecting cartilage, having a peculiar odour which has been described as a compound of the odours of old cheese and glue. The line of demarcation between the living tissue and the dead mass is a narrow hyperæmic zone. The bacillus can usually be demonstrated on the border line between the living and dead tissue.

The principal diseases caused by the bacillus are :

(1) Necrotic dermatitis or necrosis of the skin.

(2) Necrosis in punctured wounds of the foot, often resulting in quittor.

(3) Inflammatory disease of the digits in cattle (foul of the foot) and in sheep (foot rot).

(4) Calf diphtheria or necrotic stomatitis.

(5) Necrosis of the alimentary tract with secondary lesions in the liver.

(6) Ulceration of the deeper structures of the bowel wall in swine fever.

(7) Necrotic lesions associated with navel-ill.

Symptoms. — The symptoms of bacillary necrosis vary considerably according to the seat of the affection and the virulence of the organism. In many instances there may be nothing beyond the local change, while in other cases acute systemic disturbance is set up. For example, in cases of acute necrosis of the skin in the hollow of the heel and the top of the hoof in horses there is frequently intense pain, high temperature, shivering, great depression and weakness, rapid feeble pulse. In such cases secondary lesions are sometimes set up in the lungs with fatal termination. Similarly in calf diphtheria acute systemic disturbance is almost invariably set up.

Symptoms set up in other forms of bacillary necrosis are described elsewhere under their varied headings. It is therefore unnecessary to consider them in further detail here.

General Treatment. — Preventive measures should be carried out in various ways according to the particular character of necrosis to be prevented. The essential feature, however, is to protect possible seats of inoculation against invasion by the causal organism. All wounds and injuries of the hoof should be treated antiseptically as early as possible to prevent invasion by the bacillus. On farms where navel-ill and calf diphtheria are known to exist, special attention should be paid to the umbilicus at birth and to the mouth during the eruption of the first teeth. It is obvious too that whatever the form of necro-bacillosis, it is essential to separate the sick from the healthy animals, and to disinfect thoroughly everything which may have been contaminated with infective material from diseased animals.

In those cases of necrosis with external lesions the treatment consists in first removing all necrotic tissue surgically, thus exposing the deeper situations where the organism is still active. The exposed area should then be curetted and cauterised either by a hot iron or by a chemical caustic, after which it may be dressed regularly with iodine solution (1 per cent) or with carbolic acid (5 per cent). In the case of necrotic quittor where the cartilage has become invaded, it may be necessary to remove the entire cartilage surgically.

The treatment of necrosis of the internal organs is not successful. There are no known remedies of any service even in such cases where the condition is diagnosed.

G. H. W.

EPIZOÖTIC CEREBRO-SPINAL MENINGITIS

This is a contagious disease of horses with a wide distribution, being met with on the continent of Europe and in America Outbreaks have occurred from time to time in the British Isles. The effects of outbreaks are very serious, and the mortality may sometimes be as high as 80 per cent. It spreads very rapidly through large studs when once introduced. All classes of horses are susceptible both as to sex, age, breed and condition, although it is said that young and vigorous horses are especially attacked. It is most prevalent in the early summer and subsides during the colder months.

Etiology.—The actual cause of this disease is not positively made out and several organisms have been described as being the causal agent. It has been variously ascribed to a diplococcus and also to a streptococcus. The virus can be conveyed by contaminated food and drinking-water, though the immediate cause in an outbreak is usually the introduction of an affected horse into the stud. Another suggested cause is the presence of moulds or fungi contaminating food-stuffs, and that the condition is really one of forage poisoning. As against that, however, is the fact that outbreaks of the disease have occurred in studs where the food has been above suspicion. It is more than probable that the disease is essentially a contagious one.

Anatomical Changes.—The principal morbid changes are found in the brain and spinal cord, and consist of an inflammation of the membranes and sometimes of the nerve substance. There may be an effusion in the meningeal spaces and in the ventricles of the brain. Sometimes there appears to be an absence of lesions in the brain and cord. In a number of cases catarrh of the stomach and intestines has been observed.

Symptoms.—The symptoms usually commence with some degree of excitement of short duration rapidly followed by dulness and depression. At the same time there are muscular twitchings of the eyelids and ears and dribbling of the saliva. The pupils are widely dilated, the patient is in a condition of hyperæsthesia or increased sensitiveness to sounds and touch, etc. The temperature is usually elevated 2° or 3° F. while the respiration is blowing. In some instances there is also slight colic or abdominal pain. In the course of a day or two weakness of the hind-quarters is observed with a rolling gait and staggering during walking. This debility is rapidly progressive, and the patient may lie down or fall and is unable to rise and consequently struggles or fights with his fore-limbs. This is followed by stupor and semi-coma and may terminate fatally in from seven to fourteen days after the onset of symptoms.

The *prognosis* in the majority of cases is unfavourable, the disease terminating fatally in severe outbreaks in from 70 to 80 per cent. In milder outbreaks the mortality may be reduced to 10 or 15 per cent.

Differential Diagnosis.—This affection must be distinguished from :

Tetanus, in which condition there is an absence of contagion, while the muscular spasms and excitant stages are more pronounced. There is also frequently the history of a wound.

Sporadic Cerebro-spinal Meningitis.—In this condition there is no evidence of contagion, but otherwise the affection is very similar to the contagious form.

Treatment.—Treatment of this affection is not satisfactory. All excitement should be avoided and if possible the patient should be isolated and placed in slings. A purgative should then be administered, such as a full dose of aloes or calomel. This should be followed by a combination of bromide of potassium and iodide of potassium—the former as a sedative and the latter as an internal antiseptic and to assist in the removal of effusions from the brain and spinal cord. Other sedatives which may be employed include chloral hydrate and belladonna, while quinine and salicylates may be employed as febrifuges and internal antiseptics. During the comatose stage sedatives should be suspended and strychnine and arsenic substituted. There should be a complete change of the food and water supply, which may be the source of infection, and thorough disinfection of the premises should be carried out.

EPIZOÖTIC CEREBRO-SPINAL MENINGITIS IN THE DOG

Outbreaks of this affection have been described at various times. It is very doubtful whether epizoötic cerebro-spinal meningitis of the dog exists apart from the nervous form of distemper. It is just possible, however, that it may exist, especially in view of the fact that what is usually regarded as a nervous form of distemper not infrequently occurs unaccompanied by symptoms of catarrh of the mucous membranes so characteristic of distemper. Apart from that point the symptoms and general treatment are the same as for the nervous form of distemper.

G. H. W.

INFECTIOUS ANÆMIA IN HORSES

Pernicious Anæmia ; Swamp Fever

Equine infectious anæmia is a contagious disease caused by an ultra-visible virus and limited to horses and the horse tribe. Mules appear to be more resistant to it than horses. It has a very wide distribution and is met with in Europe, America and as far east as Japan, and is most prevalent in low-lying, wet districts. Infection can be transmitted experimentally by inoculation of infective materials such as blood and urine of affected animals. It can also be conveyed by the ingestion of infected fodder and litter. By passing through a series of horses the intensity of the virus becomes exalted. The common natural method of transmission is not known with certainty, but experiments carried out in Japan strongly support the view that it is transmitted by insects, particularly the Tabanidæ. Experiments carried out at the same time in Japan went to prove that the virus passes out in the urine but not in fæces.

Three types of the affection occur, viz. **acute, sub-acute,** and **chronic.**

Symptoms.—These are mainly associated with the circulatory system and the blood. There is progressive anæmia with gradual wasting and loss of condition, and the red blood corpuscles may be reduced to one-half the normal number. A prominent feature of the disease is the irregular recurrence of fever.

In the **acute form** the temperature suddenly rises to 104°-107° F. The pulse is small, weak, and frequent, and may be 100 or more, and finally become imperceptible. The heart becomes irregular and is often tumultuous and a jugular pulse may be visible. The mucous membranes in the early stages are yellowish-red but later become pallid. There are effusions into the dependent parts such as the legs, muzzle, and scrotum, which become swollen and œdematous. The patient stands listlessly, often with his head depressed and in the corner of the stable, and may take no notice of flies or attempt to remove them when they alight on him. The appetite is usually impaired or lost, though occasionally a patient will continue to eat throughout the course of the disease. The bowels are irregular and the urine albuminous. In fatal cases the debility becomes more intense, the animal drags his toes, has a rolling gait, can no longer stand on his feet and is more or less paralysed. Such acute cases may terminate fatally in from four to five days to a fortnight.

In **chronic cases,** which are usually sequel to or perhaps, more correctly, partial recovery from an acute attack, the principal symptoms are gradual loss of condition and flesh, a staring coat and pallid mucous membranes. There are occasionally attacks of fever which may last a few days or even weeks and again subside. Some degree of albuminuria is also present.

In the **sub-acute** form of the disease the symptoms take an intermediate course.

Complete recovery may occur, but in other cases animals which appear to have recovered still remain centres of infection. Recovery does not appear to confer any immunity.

Post-mortem Examination.—The only changes to be observed are those produced as the result of general anæmia, such as emaciation, loss of fat, and an œdematous condition of the tissues and cavities. The lymphatic glands are more or less swollen and juicy. Apart from these changes no gross lesions are discoverable.

Treatment. — Affected animals should be isolated at once, or if it is the first case in the stud it may be advisable to slaughter. Swampy land on which the animals have been should be drained and stagnant pools should be filled up. This procedure immediately reduces an outbreak.

Curative measures are not very satisfactory, and are devoted solely to the treatment of the symptoms. The patients should be brought indoors and guarded against exposure to rain and cold, and a course of blood tonics and internal antiseptics administered, such as quinine and iron, nux vomica, potassium iodide, and salicylates. The best hygienic measures available should be adopted and an easily digested nutritious diet provided.

G. H. W.

AFRICAN HORSE-SICKNESS (Pestis Equorum)

Perreziekte or **Paardenziekte** (*Dutch*), **Pferdesterbe** (*German*), **La Peste du Cheval** (*French*), **La Peste equina** (*Italian*).

Definition.—Horse-sickness is a disease of equines caused by an ultra-visible virus probably transmitted by a blood-sucking insect. It is likely that a non-equine animal acts as a virus-reservoir.

History and Geographical Distribution.—The first reference to a disease in horses that may be interpreted as horse-sickness was made by Father Monclaro in his account of the journey of Francisco Baro in 1569 to East Africa. Horses that had been brought over from India died and death was explained to have been due to poisoning by the natives. Of a stallion he narrates that he fell while he was led to the water and cast up some "yellow matter." In the same account, mortality occurring in cattle was likewise explained by poisoning, the poison having been placed into the grass so as to suggest plant poisoning and mislead detection. The yellow matter that was brought up by the stallion must be considered to be the pathognomonic symptom of horse-sickness, and the experience of the early Portuguese pioneers and conquerors may be interpreted as the first record about the presence of this disease in the Zambesi estuary, still deadly for horses. The term "perreziekte" or "paardenziekte," however, appeared for the first time in official documents of the Cape of Good Hope in the year 1719, when it was stated that between 1600 and 1700 horses had died before the frost arrested the disease. At that time the Colony consisted only of the districts adjoining the Cape. Years renowned for horse-sickness were 1780, 1801, 1819, 1839 and 1854–55, at intervals approaching twenty years, to which fact earlier writers drew particular attention. Such visitations were not regarded with much anxiety and looked upon "with philosophical resignation," as one writer stated. The most severe outbreak on record is that of 1854–55, when the casualty returns amounted to 64,850, but were estimated to have been at least 70,000. A census of 1854 showed the number of horses at that year to be 160,784; the mortality was thus over 40 per cent of the whole number. The disease appeared first in the eastern districts and swept westwards to the remote mountains of Clanwilliam and was reported in all divisions except that of the Cape. Definite data of rainfall for that year were only obtainable from Grahamstown and Capetown, and it was noted that the fall at the former place exceeded that of the latter by 20 inches previous to the onset of the outbreak and by 6·3 inches following it. The extraordinary heavy mortality of that year led to an enquiry being made, and a report was issued that contained the views of the cause prevalent at that time—views that are still held by many farmers in the horse-sickness districts—and advising preventative measures which, based on experience, are still adhered to now. Since that year horse-sickness apparently was no longer heard of as a plague in the south-western and eastern divisions of the Cape until 1913–1914, when it swept unexpectedly through the divisions of Uitenhage and Humansdorp and extended into the adjacent areas of George and Uniondale. In 1918 the nearest northern outbreaks of the disease were registered in the Clanwilliam division. Generally speaking, the Southern Cape is no longer considered to be a horse-sickness area. The year 1918 being an extraordinary rainy one in the northern and eastern parts of South Africa, horse-sickness was unusually frequent and carried off a great number of equines.

With the opening up of Africa it became known in many parts. The voortrekkers met it in the Transvaal and in Natal. Livingstone undertook his travels on foot or on the back of an ox. The old hunters of Matabeleland and Mashonaland knew well that only salted horses would live there. When Northern Rhodesia was occupied the same experience was noted. The first traders and missionaries and later the German farmers met it in Damaraland and what is known as South-west Africa. There is reliable evidence of its existence in the Ovambo and Angola countries and the western coast as far northwest as St. Paul de Loanda. When horses and mules were brought into Katanga by the Belgian settlers they were found to die of the disease. Reports from German as well as British East Africa, Zanzibar, and Uganda show the disease to be present there, it was also found in the Sudan along the Nile, it is reported in Abyssinia and in Erythræa as far as the Red Sea. That the disease in Katanga, German and British East Africa, and Erythræa is identical in its clinical and anatomo-pathological aspects with that of South Africa was shown in the Onderstepoort Laboratory by inoculations into susceptible horses of blood obtained from horses or mules that died in the countries mentioned. There is no definite evidence that horse-sickness is present on the other side of the Red Sea, although it is also mentioned to occur in southern Arabia. Thus horse-sickness can no longer be considered to be a South African disease, as it was thought to be at one time, but it may be called a disease of south-western, the southern, and eastern portions of Africa, leaving out so far as we know at present western and central western Africa from the Congo to the Mediterranean shore.

Topographical Conditions.—Within the areas mentioned the disease does not occur equally in all localities or in every year. It has been mentioned before that in previous times it was more frequent and much wider spread in the Cape Colony than it is now, but that, generally speaking, the Southern Cape is now free of it. It is not known in the greater portions of the Free State and Basutoland. It is but rarely found in the highest parts of the Transvaal and in the Natal highvelds. There are, however, exceptions everywhere, which are influenced by orographic, telluric, climatic, and atmospheric conditions; whilst the former remain constant, the latter are changing with every year. In South Africa it is the general experience that the low-lying parts, particularly the south-eastern and eastern coastal belt, are the permanent home of the disease. It is usually met with around lakes and pans, vleis and rivers.

Climatic and Atmospheric Conditions.—Although suitable conditions for horse-sickness may be met with in certain parts of the low country all the year round, in the greater parts of South Africa it is a seasonal plague, beginning during the summer months and increasing in frequency towards the autumn, and comes practically to an almost sudden stop with the first frost of the winter (April, May); the later the frost starts the longer the plague lasts, and cases may be seen until the end of June. The common experience has been, that nine days after frost horses can safely be turned out at grass again. The months of the greatest frequency of the disease in South Africa are February, March, and April; these are the months of the greatest rainfall. Whilst in the low veld and in certain parts of the middle veld horse-sickness during these months is met with every year to a lesser or greater extent, it appears as a plague outside only in such years as are characterized by unusual rainfalls, which lead to floods and which are succeeded by warm weather. Heavy rains at the beginning of the summer season (November and December) do not as a rule give rise to a plague; they favour an early appearance when they continue afterwards.

Special Conditions.—Given the suitable telluric, climatic, and atmospheric conditions, special circumstances are required for an animal to contract the disease. Experience has shown that horses and mules that are running free day and night are mostly attacked, but less when they are kraaled or stabled at night, and still less the better the stable, and the least when such a stable is made totally insect-proof. The practice of the last fifteen years has proved this by unmistakable results. Equines running free during the day are not subject, and repeated events have shown that it is the time between sunset and sunrise that must be considered to be the most dangerous.

Etiology.—The disease is communicable by inoculation of blood or serum of an animal suffering from the disease into susceptible ones. Microscopical and ultra-microscopical examinations have failed to show an organism in the infective material. Virulent substances passed through Berkefeld or Chamberland candles or a Pukall filter in suitable dilutions produce the disease. Hence it is concluded that the organism is ultra-visible. Attempts to cultivate it on the usual laboratory media have so far failed; its character must therefore be studied in the infective substratum, the most suitable one being the blood. The virus is found to be present in all organs. It can, however, be washed out of an organ, and the disappearance of red blood corpuscles then indicates the absence of virus (*e.g.* liver). It remains in the serum of the blood after coagulation, and is found in the serous exudate of the lungs and other parts of a diseased animal. It cannot be separated from the red corpuscles, notwithstanding repeated washings; it does not settle in the serum when centrifuged or when left standing for at least three months. It was present in the fluid of the peritoneum produced by intraperitoneal injection of peptonized bouillon; urine was found to be infective. It was traced in the blood of a fœtus from a mare that had died of horse-sickness, such blood producing disease when injected into a susceptible horse.

Susceptible Species.—Pathogenesis.—Horses, mules, and donkeys are susceptible to horse-sickness. Horses are the most susceptible; morbidity and mortality differ but little, the former attaining certainly over 95 per cent, almost 100 per cent: not all donkeys can be infected; a successful infection in these animals shows itself only as fever reaction, and no deaths so far have been reported. In mules morbidity is nearer to that of horses, if not equal, whilst mortality, although still very high, is less. Dogs can also be successfully infected. In our experiments of fifty-four dogs injected, thirty showed symptoms and nine died of typical horse-sickness. Dogs can also contract the disease from feeding on organ (liver) of infected equines. Experimentally some young angora goats could be infected developing a typical fever with no other clinical symptoms. A peculiar observation was made with goats, viz. the blood of some reacting goats proved infective for dogs and goats but not for horses, and only the strain of the virus that had passed the dog proved to be infective for the horse. The quantity of infective material that will produce the disease when injected intrajugularly or subcutaneously varies according to the substance in which it is contained and the virulency of the strain to which it belongs. Blood as a rule is infective in lower quantities than exudate. Quite a scale of viruses can be made out which vary in the intensity of their virulency. We consider the most virulent virus that which produces the disease after a very short incubative period with a very rapid course, ending practically always in death; as an attenuated virus we consider that which produces the disease after a slightly longer incubation period, with a prolonged course and leading most frequently to recovery (with our Tzaneen virus 60 per cent); of the former 0·0005 c.c. of blood may already be sufficient, of the latter 1 c.c. is not always effective, and results depend to some extent upon the place of application (subcutaneously or intrajugularly). In the case of weak viruses (Tz), doses of 5 c.c. and more have occasionally failed when injected subcutaneously, but gave positive results when injected into the jugular vein. For the purposes of practice a dose of 5 c.c. of a strong virus (O virus in our experiments) and of 5 c.c. of the attenuated one (Tz) has been adopted as a safe intrajugular injection.

Tenacity of the Virus.—The virulency of some viruses is much influenced by the age of the virus; it is gradually lost in the course of time, and by a weak virus quicker than by a strong one. As much as 5, 10, 20, or 50 c.c. old virus of the weak strain—two to three years old—failed to produce the disease, whilst the next higher dose or a dose of 100 c.c. has produced it in many instances, indicating that the pathogenic organisms may still be present. The most virulent virus preserves the infectivity for the longest, the least virulent for the shortest period. Blood may remain infective when putrid, and even when conserved in a putrid state. There are, however, certain conditions under which a non-aseptically preserved sample of blood may lose its virulency, this being due to the presence of certain bacteria, not yet definitely studied, with which blood becomes infected in vitro, a circumstance that is of far-reaching practical importance, since the infection can be transmitted from vessel to vessel by means of instruments. Blood that has been exposed to a temperature of 50° C. may lose its virulency. Carbolic acid in 3 per cent concentration does not destroy the virulency, likewise neither does a 5 or 10 per cent solution of sodium taurocholate, nor a 5 or 10 per cent solution of saponin; whilst virus mixed with 5-10 per cent lecithin solution no longer produces the disease, but does so after the two have been separated. Virus that has been dried at ordinary temperature is no longer virulent. Infective blood given per os produced the disease only after large quantities (150 c.c.) had been given.

Natural Infection.—The Insect Theory.—The observations in connection with the above epizoötic and enzoötic data find a feasible interpretation by accepting a blood-sucking insect as the disease-carrier. Attention must be drawn to the fact that at the time that horse-sickness occurs malarial fever in men is met with in the lower situated parts of the country, and that both diseases appear then under identical climatic and atmospheric conditions. This analogy has struck many observers in the Transvaal and Natal long ago. The area of distribution of the two diseases is, however, not the same; whilst both can spread, horse-sickness spreads over a much wider area and to much higher altitudes. These similar local and circumstantial conditions point to a similar mode of propagation. The observations made show in a very definite manner that horse-sickness is insect-borne, in particular the fact about the safety of horses stabled in insect-proof houses. Whilst, therefore, doubts are no longer entertained about the mode of infection, no proofs have been brought forward as to which insect is the actual carrier; anopheles cannot be accused for the reason indicated above. In order to form some idea as to the class of blood-sucking insects amongst which the carrier must be looked for, some peculiarities in connection with the appearance of horse-sickness deserve a closer attention. After heavy prolonged rain enzoötics as a rule appear explosion-like, a great number of horses succumbing to it within a comparatively short time and without there having been previously any or at least many cases. Moreover, it may then occur in places or farms that cannot be considered to be well supplied by running or even standing water acting as permanent breeding-places for mosquitoes. The transmitting insects responsible must therefore be looked for amongst those that do not require running water for their evolution and whose metamorphic cycle takes place very rapidly, the adults appearing soon after the rains. On such places or farms water may between two succeeding rains even disappear, so that it seems necessary to postulate that the carrier insect in one of its evolutive stages can maintain life in the absence of water, perhaps even resisting a certain degree of desiccation, and continue the cycle when such is again obtainable. These conditions do not obtain in the evolutive cycle of anophelinæ. Observations about the presence of mosquitoes in Onderstepoort carried out over a number of years during the horse-sickness season have given cause to suspect some species of *culicinæ* which are met with during this period (viz. Ochlerotatus, Grabhamia). The larvæ of these are found in holes made by the hoofs of horses and cattle, in which they develop into adults and the eggs are known to remain alive in moist earth for a considerable length of time ; furthermore, the metamorphic cycle under favourable conditions requires only a short period, five to six days for the larvæ and two days for the nymphæ, so that it would be completed within a week. Mosquitoes by themselves cannot produce the disease, they only act as carriers, as hosts for the ultra-visible micro-organism. It is therefore necessary that the winged insect becomes infected, and the peculiar enzoötic circumstances make it likely that the imago emerges infected, the infection having passed from the blood-sucking female through the eggs and subsequent stages. Analogies for such a behaviour are found amongst the tick-transmitted spirochætoses, where it has been proved that a number of succeeding generations remain infective. It is true that no such analogy is found in insect-transmitted diseases.

The Virus Reservoir.—The question where the transmitting insects obtain the original infection has not been definitely settled. The blood of a sick animal is infective and remains so until some time after recovery. Recent experience has shown that in some cases the blood of a recovered horse when infused in large quantities into susceptible horses can produce the disease. In one instance the blood of such a horse was infective ninety days after having received an injection of the virus. This fact may have some bearing on the question of the virus reservoir, as well as the further observation that donkeys, dogs, and goats may be susceptible. The observation that horse-sickness appears in horses introduced to countries where no equines have been before points to a non-equine as a possible virus reservoir.

Pathological Anatomy.—Three forms of horse-sickness can be distinguished, viz. (1) the pulmonary form, corresponding to the clinical form Dunkop[1]; (2) the œdematous or cardiac form, corresponding to the clinical form Dikkop ; and (3) a combination of the two.

(1) In the pulmonary form the striking lesion is the presence of a well-developed œdematous infiltration of the lungs whereby in most cases they are found in a state of inspiration and not recoiling. The subpleural tissue and the interlobular septa are distended with a clear, transparent, greenish-yellow liquid, causing a transparency of the ventral border of the organ. The thickened septa are wedge-shaped, entering from the surface into the lung tissue. They include the veins, which contrast with the transparent gelatinous envelope as blue branches. In and under the pleura of the costal and basal surfaces are large patches of gelatinous substance, flabby and doddering, giving the pleura an uneven corrugated or warty aspect, permeated or surrounded by white strands of fibrous tissue, the latter being the thicker and denser portions of the pleural covering, resisting the infiltration of the œdema. These patches can be removed by tearing the pleura off. Through the transparent part of the pleura the alveolar tissue is seen, the alveoli being sometimes fully distended. The colour of the tissue is then white or it has a reddish colour differing in intensity in the various animals according to the degree of inflation of the lung. Sometimes a mottled aspect is present; pale and red coloured patches being mingled to produce a somewhat variegated appearance. On cutting through the pleura it retracts, and liquid escapes freely or can be pressed out easily. On section through the lungs the parenchyma is found to have lost some of its elasticity. It is somewhat friable, and pressing into it with the finger comparatively lightly will break it down. It will not contract, but remains distended, and from the cut surface a clear liquid escapes or can be scraped off full of froth. In extremely inflated lungs this liquid is almost free of blood ; more of it is present when the inflation is not so intense, and in lighter cases hyperæmia may be well pronounced. Most frequently in the *apex pulmonum* and along the ventral border of the lung, particularly in the region of the cardiac notch, distinct emphysema is present. The mediastinum is likewise involved, the vessels are injected, it may be sprinkled with ecchymoses and petechiæ ; the anterior mediastinum, including the peritracheal and perivascular connective tissue and the lymph nodes, are thickened by the gelatinous infiltration, particularly at the *apertura thoracis*. Where the pleura is reflected on to the larger vessels and in the hilus of the lungs liquid escapes copiously on cutting, and likewise in the posterior mediastinum, between the aorta and œsophagus on the one side and the œsophagus and the dorsal border of the lungs on the other side, including the mediastinal lymph nodes. The bronchial lymph glands are likewise enlarged and very soft in consistency. The pleural cavity as a rule contains an increased quantity of clear fluid, which may amount to several litres. The costal pleura is generally not much involved in the process. Larynx, trachea, and bronchi are

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[1] "Dunkop" means "thin head," "Dikkop" means "thick head"—thin to emphasize the non-appearance of swellings.

usually filled with a frothy substance or with a yellowish liquid that forms a fibrinous thready coagulum only in rare cases. The vessels of the pericardial pleura may be injected and some œdema may be present in the base of the pericardium, the parietal serosa as a rule is smooth, moist, and glistening. No increase of liquid is noted in the pericardial sac. The epicardium is most frequently normal, occasionally there are ecchymoses and petechiæ bordering the transverse and longitudinal grooves. Blood is usually present in the right ventricle, sometimes it is badly coagulated. Under the endocardium of the septum and the wall of the left ventricle suggillations, ecchymoses, and petechiæ are frequently found; they are rarely absent in the musculi papillares. The myocardium usually shows no changes, it is of normal colour, moisture, and transparency in its superficial surfaces. Occasionally a hæmorrhage is found within or near the endocardium. The stomach usually contains food. There is frequently a fairly viscid mucous deposit in the fundus and in the pylorus. The mucosa of the former is practically in all cases found diffusely red or patchy red, the patches being of different intensity or distinctly hæmorrhagic with extravasations of blood in some parts. The mucosa may be slightly thickened. The mucosa of the small intestines most frequently shows no lesions, but sometimes a patchy reddish discoloration may be present. A diffuse reddening with slight swelling of the mucous membrane is present in the large intestines, an œdematous infiltration is traceable in the submucosa and in the lymph nodes. Under these conditions some liquid may be present in the abdominal cavity. The contents of the large intestines are usually soft. The liver is frequently found rich in blood, the borders slightly rounded, the colour dark, the parenchyma moist and fairly friable, blood escaping from the cut surface. The lobulation is usually distinct; fatty degeneration is rarely present. The spleen is usually of normal size, it may however be found increased, with the pulp somewhat softened. The kidneys most frequently show hyperæmia of the cortex. The urine is generally of normal appearance.

(2) In the Dikkop form the striking lesions found are in the subcutaneous and fatty tissue of the *fossa temporalis*, which is found bulged out; in that of the region of the masseter; of the mandibular space; and of the parotid region. There we find the presence of the œdema in the lymph nodes, in the parotid and submaxillary glands, in the areolar tissue filling the spaces around the pharynx and larynx. It is also present in the connective tissue of the neck, and along the jugular groove, extending into the *apertura thoracis*, under the skin of the shoulder and withers, in the subcutaneous tissue under the shoulder and in the axilla, along the back; and in cases of some standing it is found in the sternal region and along the abdomen, and on the arm and forearm. On cutting through the muscles of the head and neck yellowish liquid is found in the fasciæ and aponeuroses separating or enveloping the muscles in the various regions mentioned and even in the deepest layers along the cervical vertebrae (musculi transversales). It infiltrates the muscular tissue, which appears moist, the colour of the muscles being somewhat darker than usual, frequently of a brownish tinge. The mucosa of the mouth, tongue, and pharynx may be bluish coloured (blue tongue), the intermuscular tissue of the tongue is sometimes œdematously infiltrated, and the organ shows an increased volume. The submucosa of the trachea is usually thickened by yellowish fluid and gelatinous exudate, which is present in its dorsal portion. The pericardial sac is practically always distended with liquid, the quantity averaging about half a litre in the majority of cases, but reaching as much as one to two and a half litres and more in extreme cases, which are by no means so rare. The liquid in a freshly made post-mortem examination is clear and has a yellow to brownish colour. The base of the pericardium may show slight œdematous infiltration as well as that of the heart; the pleura of the pericardium is injected. The parietal serosa is usually smooth and glistening. The epicardium frequently appears diffusely brick red or patchy red, in particular around the circular and longitudinal grooves; also the auricles may be diffusely red. This red colour due to extensive hæmorrhagic extravasations is also found in the subjacent musculature of the myocardium, which it may permeate, forming extensive suggillations. The endocardium of both ventricles shows diffuse or patchy red discoloration due to extravasations of blood underneath. The heart is found in diastole, the right ventricle much distended containing a badly coagulated clot, sometimes separated into cruor and plasma. The lungs are most frequently in the state of expiration, normally retracted, the pleura even shrivelled, the lung tissue of normal consistence, but darkly coloured, fairly rich in blood. Very slight pulmonary œdema may be present. A slight hydrothorax may be found, but is rare. The liver as a rule is enlarged, rich in blood, with rounded borders, dark in colour, the lobulation is usually distinct, the cut surface sometimes of nutmeg appearance, the central veins distended and surrounded by an opaque zone, the consistency friable. Fatty degeneration may be found in various degrees of intensity. Swelling of the spleen is frequent, the dark pulp obscuring both follicles and trabeculæ.

Stasis of the kidney is observed, frequently accompanied with a cloudy swelling and fatty degeneration. The lesions in the intestinal canal correspond with those found in the pulmonary form of the disease.

(3) In the mixed forms both pulmonary and cardiac lesions as well as the œdematous infiltration may be found together. They form the minority of cases.

Symptomatology.—Clinically we may distinguish four different forms, viz.: (1) horse-sickness fever; (2) the pulmonary form (Dunkop); (3) the œdematous or cardiac form (Dikkop); (4) the mixed form.

(1) The **horse-sickness fever** is the one form that is difficult to diagnose under the conditions of practice. It offers no pathognomonic symptoms that would allow its differentiation from any other fever, except perhaps the typical course. Subsequent to the inoculations of viruses of known attenuated virulency the fever is regularly noted in susceptible animals, and in particular in mules and horses that have been submitted to the horse-sickness immunisation. It is the form found in donkeys. Under these conditions fully 60 per cent of the horses or mules only show the fever reaction. It begins with an incubation period, which depends upon at least two conditions, viz. the virus and the animal injected. An attenuated virus (*e.g.* the virus Tzaneen of the Onderstepoort experiments) will frequently only produce fever in a susceptible animal. It has an average incubation period of five to seven days, but shorter periods of less than five days and longer ones to fifteen days, but rarely up to one month, may be met with. Similarly the incubation periods vary in the fever produced in immune horses injected with a virus that may break the immunity. The onset of the reaction is not sudden, the first rise differs but little from the temperature of the previous day, it continues to rise the next day to a point higher than the day before, and does so during the first four to five days until the acme is reached, when a descent will take place in like manner until the normal is reached again, viz. twelve to fourteen days after infection. The fever is thus a typical one and of a remittent type, morning remissions and evening exacerbations differing about one to three degrees. The acme may reach anything from 102° F. to 106° F., and it may even for two or three days remain at that height, usually it averages 103·5° F. to 104° F. It may occur as soon as the ninth or tenth day, but usually about the twelfth or thirteenth day after infection. The *stadium incrementi* lasts frequently a day or two longer than the *stadium decrementi*, and in place of a gradual lysis a descent resembling a crisis may be noted: this is, however, rare; the course of the fever lasts five to eight days, it is rarely protracted. The fever in the majority of cases is not accompanied by other symptoms. The conjunctiva is usually normal, rarely slightly injected. The pulse may be increased in frequency, 40 to 44, very seldom to 50, and may be perhaps a trifle fuller. No changes are noted in the respiration which would attract attention, and the frequency counted in a number of cases averaged 12 per minute. A certain degree of anorexia may be present, more pronounced in some horses than in others, and not always in direct relation to the height of the fever. If present it is usually noted during the latter half of the fever curve. No changes are noted in the distribution of the surface temperature. Practically no disturbance in the general demeanour is present, except perhaps a little depression after the acme has passed, the horse showing dulness whilst standing in the stable.

(2) The **pulmonary form** (**Dunkop**) may be considered as the fulminant or peracute form of horse-sickness. It is met with in horses and mules and in dogs experimentally infected. In equines as a rule it follows the injection of an exceedingly virulent virus (*e.g.* ordinary virus in the Onderstepoort experiments). It is noted rarely after the injection of an attenuated virus (*e.g.* low generation of Tzaneen). Generally it occurs after a short incubation time that after inoculation may be as short as two days in rare cases, oftener three days, and most frequently four to five days, in rarer cases longer. Experience has shown that the shorter the incubation period the shorter the course of the disease itself, the longer the former the more prolonged is the latter. The first symptom is the rise of the temperature similar to what has been described under horse-sickness fever. It rises here, however, very quickly, frequently within three days, to the acme, which in most cases is high, reaching 105° F. to 106° F. The *stadium incrementi* may sometimes also last longer, four or five days, when either the ascent of the temperature is still gradual until the acme has been reached on the last day or the temperature of the acme has been reached earlier and remains for a couple or more days at the same height. The fever in the greatest number of cases is still in the acme when the horse dies. During the first and second days after the onset of the fever no other clinical symptoms are registered, the pulse rate is but little affected, and the horse's general demeanour does not draw the attention of the attendant. The first symptom to be noted is the accelerated and impaired respiration, a polypnœa that either increases gradually or begins almost suddenly and reaches a maximum within a very short time. The dyspnœa is of both inspiratory and expiratory character. Both wings of the nostrils at first

move quickly, later the nostrils are fully distended. The larynx moves rapidly up and downwards, and the head and neck are slightly stretched. Nodding is particularly noticed in the movements of the ears, which are partially or totally drooping. Sweating takes place at the side of the nose, the base of the ears, and later on various parts of the body, as the anus, inguinal region, flank, side of the neck, etc. The eyes are staring and reveal anxiety. The outlines of the muscles of the face and of the grooves between them appear somewhat sharp. The number of respirations soon reaches 40 to 60 per minute, but may pass 80 and 100. The respiratory movements, at first somewhat superficial, become gradually more pronounced in both the chest and the abdominal wall. The ribs are seen to make wider excursions. Immediately following the contraction of the chest that of the abdominal wall takes place, thus giving rise to a well-pronounced groove along the *arcus costarum* or costal ridge. (This double respiratory movement is considered pathognomonic by the farmer.) Inspiratory and expiratory symptoms of a graver nature appear during the further course. The animal does not lie down, the front legs are spread and a rocking movement forwards and backwards of the body takes place, the back is slightly arched, the tail nodding, and the anus and the paralumbar region moving in and out, the mouth occasionally kept open and tongue slightly protruding. In the dog pronounced labial breathing is present. Soon after the onset of the polypnœa coughing appears in most animals, it is vigorous and dry at the beginning; a number of impulses occur in rapid succession, amounting at times to real fits. These coughing fits increase in frequency and stand in relation to the dyspnœa noted. The presence of a whitish-yellow streak down the upper lip, viz. dried serous discharge, is a symptom sometimes seen at an early period and before or soon after the coughing has started. Coughing and discharge stand in relation to each other. At the beginning the fits of coughing are short and dry and occur at longer intervals; later they last longer and occur at shorter intervals, they are moist and gurgling and bring up an increased quantity of frothy yellow liquid, and towards the end of a paroxysm or immediately following it streams of it escape with great force, passing through the nose. The cough is frequently succeeded by deglutition. When choking is approaching the horse becomes more and more restless, it moves about, sometimes it lies down, but soon and quickly gets up again; when choking is overtaking the horse, during and after fits of coughing, it sways about, it staggers, head and neck are thrown up and down or sideways, discharging yellow foam and liquid abundantly, and finally the animal falls down and lands on its side or on the sternum, where it rests with all four legs drawn to the body, dying with the neck and head bent downwards leaning against a corner of the stall. When landing on the side and dying, head and neck are stretched out, a period of apnœa follows a few deep inspirations at prolonged intervals and dilatation of the pupils. Sometimes the animal dies with the first discharge; sometimes it dies without having shown any; but invariably a short while after death foam will be noticed in and in front of the nostrils. The horse is actually drowning in its own serum. There are cases on record where horses or mules have recovered after copious discharge of the mucous foam has been noted, bucketfuls having been voided, but these observations are exceedingly rare and represent exceptions.

With the onset of the polypnœa exaggerated vesicular breathing is noted; it is present during both inspiration and expiration. With the appearance of the hypochondriac groove crepitant râles are heard in the ventral region of the lungs, first a fine crackling noise towards the end of an inspiratory movement, later and higher up moist râles make their appearance, which at times may be very coarse and of different descriptions: gurgling, bubbling or whistling, and wheezing. Pleuritic friction sounds are frequently noted as grating or rubbing sounds usually about the centre of the chest wall. There is finally mingling of all these sounds into one indefinite chaos. Moist râles and gurgling noises are heard in the trachea at about the time when coughing has set in. The period from onset of the polypnœa until death varies in the different horses; it may be very short, hardly half an hour, but it may last some hours and even one day, but rarely more. *It frequently happens that a horse placed under the saddle or inspanned from all outward appearances in normal health may suddenly develop the acute symptoms and die within a quarter of an hour to half an hour.*

The conjunctiva during the first stages remains normal, later it may be diffusely reddened, sometimes it becomes ecchymosed. Cases that pass over into recovery, with the few exceptions already alluded to, do not show such a rapid and violent course; the dyspnœa develops gradually and the polypnœa does not reach the maximum number of respirations. Coughing in these cases, when present, is not so frequent, and either does not lead to discharges of froth through the nostrils or else only to that of a small quantity of liquid, although crepitating noises and râles are noticed on auscultation. In cases of recovery equally high temperature is recorded as in cases that end fatally, which from the acme descends to normal in the lytic manner described before in the horse-sickness fever. The curve of a pure pulmonary form when recovery takes place thus shows a high acme and is extended in length (ten to fourteen days), the *stadium decrementi* lasting frequently as long as the *stadium incrementi*. A slight dyspnœa may still be present at a time when normal temperature conditions prevail again.

The pulse at the beginning of the fever reaction shows hardly any appreciable changes, and none, or only very little, during the evolution of the fever. It hardly increases in number, about 40-44 per minute, the beats are regular and of equal volume, it becomes somewhat fuller and stronger. With the onset of the dyspnœa these conditions then change somewhat, the frequency increases to 50 or 60, the quality becomes somewhat softer, and when the frequency is still increasing even weak and thready.

The appetite remains good during the prodromal stage, only occasionally the horse may show some anorexia, even at the beginning of the polypnœa the horse may be seen to be feeding well. Sometimes it is noted when the dyspnœa is distressing that the horse grasps the food hurriedly, chewing for a while, and then suddenly abandoning it, to return again and do the same. Even at the time of the greatest distress some horses will grasp some food and will die with such between the lips and teeth. On the other hand, some horses from the first onset of the dyspnœa will not touch food or water. Sometimes horses are heard to neigh with a moist voice during the period of asphyxia. Generally the defæcation that takes place during the prodromal fever and the subsequent polypnœa is normal; in some cases, however, copious evacuation of abnormal softness takes place in the later stages. Micturition is noted to occur during the progress of the asphyxia and urine is repeatedly voided in jets.

Recoveries from Dunkop are rare in comparison to those from Dikkop. Under the conditions of practice Dunkop is rarely noted before the dyspnœa has set in. Anorexia and high fever during the horse-sickness season suggest the presence of horse-sickness.

(3) The **œdematous, Dikkop, cardiac,** or **acute form** has its name from the striking swelling of the head extending to the neck and body and the serious affection of the heart. Dikkops are frequently noted in susceptible animals after the injection of attenuated viruses and usually in equines that break down in immunity (natural infection). When a virus is of a very virulent nature, then practically all horses that have not shown the pulmonary form will develop the œdematous form and the majority will die of it. The more deaths of Dunkop type such virus produces, the more certain is the death from Dikkop in those that develop this form; the less Dunkop cases it produces, the more Dikkop cases ending with recovery will appear. It begins with an incubation period, the length of which in the first instance is dependent upon the virus injected; if the virus is a weak one (*e.g.* Tzaneen virus), then the incubation period is usually longer; if it is a virulent one, it is generally shorter. The shorter the incubative period, the quicker the evolution of the symptoms. The incubation period averages usually five to seven days, rarely less; it may be any length of time up to a month, but very rarely longer; the longer the incubation period lasts, the more frequently Dikkop is observed. The fever curve that ensues resembles the one described under the horse-sickness fever, the exacerbations keep within very moderate limits in most cases, in some hardly any deviations from the normal excursions are seen; indeed, the course of Dikkop is most frequently characterized by a relatively low fever as compared to that in Dunkop. The acme in the majority of cases is reached about the twelfth or thirteenth day, and is succeeded by either a lytic, a slightly protracted crisis, or a critic descent; the latter most frequently the precursor of a collapse, the former two are frequently succeeded by a slight subnormal temperature recorded for one to two days. Hence the ascending branch of the fever curve is generally longer than the descending one. The duration of the fever period lasts about eight to ten days, so that by the fifteenth to seventeenth day after infection the return to normal temperature has taken place. The average temperature will be 102·5° F. to 104° F. The symptoms of Dikkop, viz. the œdematous swellings, appear as a rule when the fever acme has passed, viz. about the fourteenth or fifteenth day after virus injection. If they appear before that time, then the case has a very rapid course and ends most frequently with death; the swellings increase in extent and severity as the fever disappears and even after it has disappeared. Its evolution may be either very rapid, protracted, or comparatively slow. The first indications are a filling up of both temporal fossæ until they reach the level of the bones. These may be the only swellings that ever occur, and they soon disappear, lasting usually one to three days, rarely longer. Usually, however, the swellings bulge out and stand out prominently; they may reach a maximum within a day, but more frequently only after two to three days; they remain so for two or three days, and then gradually disappear, remaining for a week to ten days, rarely longer, and then of course are still present at a time when all fever has disappeared.

Contemporaneously with the bulging of the *fossa temporalis* swellings appear of the adjacent temporal region and the temporal muscles stand out prominently like a cushion, the swellings may also go over the supra-orbital ridges into the upper and lower eyelids, which appear tense, the wrinkles disappearing. The upper lids droop, the lower lids rise, and so finally a complete closure of the eyes is effected. Mucous discharge may be present. In some cases and not so infrequently the œdematous infiltration causes a turning out of the lids, the swollen mucosa then appears glistening and deeply reddened, discharging a sanguinolent serous liquid. The closed eyelids cannot be moved or only very slightly. This state of affairs remains for at least two to three days and longer. With the increase of the swellings in the *fossa temporalis* it is noted that the eyeball is also apparently impaired in its movements; it is slightly protruding, a somewhat staring look is developing, the cornea losing some of its lustre. Most frequently the bulging of the temporal fossa is succeeded within a short time, usually on the same day, by a swelling of the masseters, the ventral portion somewhat projecting sideways so that the head assumes a square appearance; the swelling also extends to the mandibular space, which becomes filled, so that the lymph nodes and artery can no longer be felt through the skin. In rarer cases the swellings extend to the buccal region, to the lower lips, the upper lips, and the tongue, which may then stick between the teeth, whilst the lips somewhat rigidly stand off the gums; the nose also is rarely involved, the *alæ* being somewhat rigid. Indeed under exceptional conditions the whole head has a swollen appearance. With the increase of the swellings in the head and usually subsequently, swellings of the neck occur. They are first noted in the parotid region, and extend dorsally on to the occiput, on the atlas and axis, ventrally into the laryngeal region, and finally along the sides of the neck. The jugular grooves fill up, all wrinkles disappear, and the ventral border of the neck has a rounded appearance. Both head and neck are now kept somewhat stretched, the angle between mandible and neck is increased according to the degree of swelling present. When the swellings are very extended there is only little movement noted, head and neck and forequarters are moved as a whole. The swellings may also appear in the shoulder and fill the whole curvature between the side of the neck and the scapular region; they may go over on the side of the withers, behind the shoulder, and along the back. They are rarely under the breast from the beginning. Horses that show extensive swellings most frequently die; recovery is, however, also possible in cases where they give cause for the greatest alarm. When recovery takes place they disappear, the œdema diminishing gradually on the head and neck at about the same time; the temporal region is usually the first part to be free again. Some of the œdema sinks down the neck, so that the clavicular fossa and the breast, as well as the sternal, epigastric, and sometimes the mesogastric regions, are swollen. It finally goes over into the arm and forearms and down the legs. Both forelegs become greatly thickened, causing a stiff gait. Finally it disappears completely. In the beginning all swellings are tense to the touch, impressions disappear directly after removal of the finger; in later stages they remain. The temperature of the swollen parts is that of the body, at least not subnormal, and manipulation is not painful. Only on some rare occasions on the occipital region, extending laterally not below the wings of the atlas and caudally not beyond the axis, a hot painful swelling was noted, the horse then stretching the head almost straight out and resenting any attempt to touch it. The conjunctiva at the beginning of the fever shows no alterations, later it may become injected; ecchymoses may or may not be present. Usually a diffuse reddening appears, in some cases of a bright colour, in others it is dark red to bluish. Generally speaking, an early diffuse and purplish discoloration of the conjunctiva is an alarming symptom. Frequently at the same time a bluish colouring of the mucosa of the mouth is noted; it indicates a serious prognosis and in the majority of cases it precedes the approaching death (blue tongue).

The pulse during the onset of the fever reaction is perhaps slightly stronger and fuller than normally, but its rate is not yet increased. Approaching the acme it increases very slightly; it may rise from 40 to 44 or 48, and may remain at this figure during the descending portion of the fever curve. Usually when the acme is passed and the symptoms of Dikkop begin to appear the quality of the pulse alters, it becomes softer and weaker and thready, almost imperceptible or wholly so. Then the rate increases to 50 and 60, rarely more. Sometimes the rhythm is also altered; dicrotic or deficient pulses can be registered. The increase in rate and the weakening of the beats stand in some direct relation to the severity of the swellings present. The heart action also undergoes alterations when the symptoms of Dikkop are developing; generally speaking, the area of impulse increases, although the impulse is rarely so pronounced as to shake the body wall; in some horses it is distinctly noticeable by hand, in others only faintly or not at all, *i.e.* it may disappear during the course of the disease, to appear again when recovery has taken place.

The sounds, at the beginning strong and distinct, become during the course frequently weaker and diffused. The respiration during the ascent of the fever shows no changes in frequency or quality, and in a great number of cases that show the Dikkop symptoms only mildly interference is never noted. Frequently, however, the respirations increase in number and become distinctly abdominal, particularly in cases that end fatally. Generally the frequency remains below 30 per minute; the groove along the costal arch becomes well pronounced and the double movement of the abdominal wall is distinct; the costal movement is, however, less prominent; these symptoms diminish with approaching recovery, viz. when the œdematous swellings are disappearing or have disappeared, but they increase with the increase of the swelling and stand in a certain relation to the impaired heart action. When the excursion of the abdominal wall becomes more accentuated the groove deepens. A sudden increase in the respiratory movements of a pumping character indicates that the horse is approaching death. In mild cases animals remain most frequently standing throughout the disease, or when down take up the sternal position and rarely stretch themselves out flatly. Only in rare cases they remain standing until shortly before death, sometimes resting the head on the manger, with drooping ears. Usually horses or mules go down with the increase of Dikkop and remain in sternal or lateral decubitus, or take up one or the other alternately. Sometimes previous to lying down they show much restlessness; they walk about in the box, turn suddenly round, go up and down, change from sternal to lateral decubitus, groaning frequently when changing the position. At first lying down is carefully carried out, later the animal lets itself drop. This state of affairs may last but a short while, when the animal will die, or it lasts for one or more days. So long as the ears and legs remain warm the prognosis is favourable, with their cooling death is approaching, cold perspirations may then also be present. Tremor of muscles of the shoulder and hind-quarters are then frequently seen. Sometimes, and in cases where the swellings are not so extensive, death approaches even more alarmingly. The restlessness can now be described as colic, the animal goes very frequently up and down, and when down may roll over on its back to rise up again; it will paw with the forelegs and show spasms in the hind legs. When standing excessive dyspnœa is noted, almost as strongly pronounced as in Dunkop, but without discharge from the nostrils. In some rare cases the animal remains standing most of the time whilst the dyspnœa increases rapidly, the respiratory movements developing into a continuous pumping of the abdomen, shaking the whole body. The animal will suddenly stagger and fall and expire shortly afterwards. During the ascending portion of the fever the animal's general demeanour remains normal; it is attentive and the eyes are bright; the temperament becomes affected as soon as the œdema grows in intensity and extent. The animal is quiet with head slightly hanging and ears drooping; when the swellings enter into the eyelids it looks distinctly sleepy, and the more the swellings are pronounced the greater the dulness; the head may then be hanging to the ground in an almost straight line with the neck. Loss of appetite is noted when the fever is approaching or has reached the acme, and complete anorexia is usually present during the succeeding lysis, even when the fever is of a very light type and the swellings only moderately developed. The abdomen has then a tucked-up appearance. Under the conditions of practice the Dikkop form is usually noted with the onset of the swellings, rarely the presence of anorexia draws the attention before their onset. From the above description it follows that an animal with Dikkop is in the latest stage of the disease.

(4) The **mixed form** of the disease is rare in comparison to the former two, and may be found in an animal suffering first distinctly from a pulmonary form, viz. the pulmonary trouble not yet expressed in an alarming manner, at a time when the fever has reached its acme, when suddenly swellings of the temporal fossa will appear; they are usually moderate and rarely show any marked extensions, the animal most frequently dying soon after their appearance, rarely lasting longer than one or two days. In the second form the reverse sequence takes place. An animal that is showing all the lesions of a Dikkop, usually not to a severe degree, is suddenly carried off by a sudden attack of the pulmonary form that in no way differs from the one described before. The end may occur during the descending portion of the fever curve, when the lytic descent of the fever will suddenly show a rise again. The mixed form of the disease is frequently the expression of a double infection, as has been clearly brought out in experiments with subsequent injections of two viruses at short intervals. Under the conditions of natural infection the mixed forms are also noted.

Complications.—These may be divided into two groups, one connected directly with the disease and one in which the complication is a secondary occurrence.

To the former belongs **paralysis of the œsophagus.** In particular it may occur in horses that are suffering from a severe and usually early Dikkop, but it has also been noted when the swellings were not much marked.

The first symptoms are regurgitations through the nose. Sometimes attention to its existence is drawn by the presence of masticated food in the manger or on the floor or smeared against the walls of the loose box. When water is given to the horse it will as a rule greedily drink it, but all water and with it food materials will return through the nostrils. The paralysis may be complete and all liquid will return through the nose, or it may be partial, when some of it will pass down. In the former case deglutition is not succeeded by the passing of the bolus down the œsophagus. On examining the left jugular groove, no movement in it is visible. On palpation deeply into the groove the stuffed œsophagus is felt as a firm thick rope-like structure. This state of affairs may last for some time; in horses recovering from horse-sickness it may still be present when all the swellings have disappeared. In its course the mucosa of the nostrils becomes diffusely reddened and an offensive odour is noted, the discharge is no longer food but is most frequently frothy mucus. Such horses lose rapidly in condition, the abdomen is tucked up and the eyes are soon sunken, an increased abdominal respiration shows the presence of a pneumonia to which the horse succumbs, if not killed before. In some cases recoveries take place, and this after treatment by massage and blistering of the jugular groove when first the deglutition of water becomes possible again and later that of solid food. We have also noted a temporary recovery ending finally with complete paralysis, whilst a partial recovery was present for about four days before the paralysis had completely disappeared. On post mortem of horses that succumbed or were killed the œsophagus was noted to be stuffed with well-masticated food-stuffs usually in its total length, the stuffing being so tight that after splitting the œsophageal wall the contents dropped out as a sausage-like body, more or less solid according to the age of the disorder, the stuffing being sometimes almost dry. In some cases only parts of the œsophagus were filled with foodstuffs, always those nearest the oral opening, whilst the lower part was found empty. The lesions in the lungs are those of a gangrenous pneumonia

As a very rare occurrence, only once seen by us, may be registered a **paralysis of the nervus opticus** showing itself as an amaurosis of both eyes subsequent to a recovery from a severe Dikkop.

Colic has already been described as a symptom of horse-sickness in some instances, sometimes indeed it may be present before the acme of the fever and may pass off again, or more frequently only occur in the latter stages of the disease. In several cases treatment with chloral hydrate removed the pain and horse-sickness then took its usual course.

Of the diseases indirectly connected with horse-sickness **Nuttalliosis (Biliary fever)** must be mentioned. It is sometimes noted in horses that pass through an otherwise mild attack of the disease, it frequently forms a complication in the immunization of horses. Its presence shows itself in a disturbance of the fever curve by exacerbations that arise suddenly and usually reach a high degree; a rapid increase of the pulse also takes place. Pale and yellow conjunctiva will reveal the presence of an anæmia accompanied by icterus varying in intensity. Microscopical examination shows the presence of the parasites.

Differential Diagnosis.—In South Africa there are two fevers met with in horses that may lead to mistakes in the diagnosis, particularly in experimental work, viz. **Ephemeral fever** and **Spirochætosis.** Both have shorter incubation periods and a shorter course and usually a high acme. Anorexia may be present in both, but other clinical symptoms are absent. The latter disease can frequently be diagnosed microscopically, when the presence of *Spirochæte theileri* can be found. Neither ends fatally. **Acute biliary fever** is frequently mistaken for horse-sickness, particularly when dyspnœa is present. The pale yellow (lemon) conjunctiva and the accelerated pulse allow clinically a definite diagnosis; in doubtful cases the demonstration of *Nuttallia equi* settles the case. Acute pernicious anæmia or the paroxysms in the course of this disease frequently give cause to suspend diagnosis for a day or two. When typically pronounced, the orange-tinged, ecchymosed, pale conjunctiva is characteristic.

The Nature of Immunity in Horse-sickness.—A horse or mule that recovers from an attack of horse-sickness, Dikkop or Dunkop, is called in South Africa a "*salted*" animal, and is highly valued owing to the fact that recovered horses show a considerable amount of immunity and that recoveries only occur to a very small extent. The term "salted" refers also to horses that have not been noticed to show horse-sickness, but have lived in localities known to be fatal to horses. We now know from our experimental results that horses may "salt" from this disease without ever showing any symptoms except a slight fever, which of course is overlooked in practice. We are not in a position to state definitely whether there are animals that are naturally not susceptible to horse-sickness, although in experiments we met with horses that could not be infected. There is no doubt that there exists an individual resistance that varies with quality and quantity of the injected virus, a phenomenon that probably is more connected with the particular virus than with the idiosyncrasy of the horse. Experience shows that to a highly virulent virus (*e.g.* the "Ordinary" virus used by us for the purpose of hyperimmunizing) there are only exceptional horses that will completely resist and equally few that will recover, whilst to a virus of less virulency (*e.g.* the virus "Tzaneen") more will resist and more will recover. These points have been mentioned before, and are repeated here to show that resistance and recovery are apparently dependent upon the quality of the infecting virus. Resistance to horse-sickness under natural conditions may be due to three contingencies: either the exposed horse is not bitten by the virus-transmitting insect; or the injected virus is not virulent enough to cause the disease; or the horse is completely immune to any virus. The first contingency most probably depends entirely upon the number of infecting insects that may be about. It has been noted in some of our experiments that of a certain number of susceptible horses exposed to natural infection the greater portion died, whilst the surviving ones when tested on immunity succumbed. It is difficult to understand why not all horses become infected; it may be that some horses attract the biting insects more than others. In a very bad season or locality no horse exposed will escape infection.

The question of immunity following recovery is a very important one, and was a very disputed one because deaths were frequently noted after alleged recovery. Dunkop (pulmonary form) is frequently confounded by the layman and the tyro with other diseases that may show symptoms of dyspnœa. In cases of Dikkop such mistakes occur less frequently, hence the conclusion that horses recovering from this form are better "salted" than the former, meaning by the word "better" that fewer horses were observed to die after the latter diagnosis had been made. That salted horses can contract the disease again is an old observation; it is called by the Boers "aanmaning," which is translated as "relapse," both terms implying that the horse is suffering from a true relapse, as observed in human malarial fever. Such a relapse does not take place in horse-sickness, and all outbreaks in immune (?) animals are new infections. We speak of them as *breakdowns in immunity*. The earlier observers had already established the fact that such breakdowns end in the greater number of cases with recovery, and that death is frequently due to adverse exposure and treatment the sick horse or mule had been exposed to. But nevertheless it has also been found that the protection conveyed by recovery varies in the different years, and in bad seasons salted horses die more frequently, a phenomenon that is laid stress upon to emphasize a particularly bad year. The question of immunity has been investigated by us experimentally on a large scale, both by testing animals that have recovered from horse-sickness, either by means of inoculation or by exposure to natural infection. In the year 1905–6 a method of immunization of mules was introduced by us, and that year 3235 mules were inoculated and exposed to horse-sickness. Accurate records were kept for the first year, and the result was that 45 mules were reported to have shown breakdowns with subsequent recovery and 21 breakdowns with death; the total number of breakdowns thus amounting to 66 mules = 2 per cent. In 1914–15 on the farm Onderstepoort—a reputed bad farm for horses—101 immunized horses were exposed, of which number 16 horses showed breakdowns of immunity, 6 recovered, 10 horses died with symptoms of Dikkop, viz. 16 per cent breakdowns, 10 per cent deaths. In the year 1916–17 in the same place 224 immunized horses were exposed and 14 breakdowns were noted, 6 horses recovered and 8 died = 6 per cent breakdowns and 3·5 per cent deaths, all with symptoms of Dikkop. In the same season 50 immune horses were exposed in Zululand—a notoriously bad locality—and 4 horses died of horse-sickness = 8 per cent, the diagnosis being confirmed by subinoculation. The number of recoveries was not stated. In 1917–18 a total number of 208 immune horses were exposed; of these, 64 breakdowns were noted = 30·7 per cent, and 19 deaths = 9 per cent. It is of interest to note that of the 64 breakdowns 62 showed Dikkop and only 2 Dunkop. These exposure experiments should indicate to what extent breakdowns can occur in immune horses generally, not taking into consideration the quality of immunity and the degree, both distinctions to be made in the interpretation of experimental results obtained from the inoculation of different viruses and of different horses. Although no figures are at our disposal to show in what proportion naturally salted horses break down and to what extent recoveries take place, we must assume that the figures given above represent a similar state of affairs, since all immunized horses pass through an attack of horse-sickness that in all other respects resembles this disease naturally contracted. With reference to the degree of immunity we distinguish that in horses recovering from an attack from that increased by hyperimmunization, for which purpose the horses usually receive an amount of not less than 10 litres of virulent blood three times in succession at intervals of six weeks to two months. That there is a difference in the degree of immunity is shown by the fact that the hyperimmune animal produces a protecting serum, whilst the simply recovered horse or mule does not. Of the 101 horses exposed in Onderstepoort, 1914–15, 25 horses were hyper-immune, and of these 3 died = 12 per cent; of the 224 horses exposed in 1916–17 in Onderstepoort, 79 were hyperimmune, and amongst these 5 breakdowns occurred = 6 per cent, of which 2 were fatal = 2·5 per cent. Of the horses exposed in Zululand, 23 were hyperimmune horses and 2 died = 9 per cent. Of the horses exposed in 1917–18, 9 were hyperimmune and 1 showed a breakdown with recovery = 10 per cent. The total number of horses exposed in the different years amounted to 583; of these, 447 were immune and 136 hyperimmune, of the former 86 (19 per cent) showed breakdowns with a mortality of 33 (7·3 per cent), of the latter 11 (9 per cent) showed breakdowns with a mortality of 7 (5 per cent). There is thus a slight difference which might not be noted if equal numbers of horses of both categories had been exposed. The fact to be emphasized is that even a highly immunized horse is not protected against a natural attack of the disease. The differences in morbidity noted in the various years stand most likely in relation to the infection present and form an index of the severity of the season.

As far as the *quality of the immunity* is concerned it can be stated that it does not stand in any relation to the severity of the clinical disease from which the animal has recovered. In this respect Dikkop must be considered to be a more severe form of horse-sickness than the horse-sickness fever. Statistics compiled from the figures given above show that of 90 horses that showed breakdowns with Dikkop, 58 had had a previous Dikkop (from inoculation) = 64·5 per cent, as compared to 32 horses = 35·5 per cent, that had previously only shown the horse-sickness fever; and of the 34 deaths that occurred from relapses, 28 horses (82·4 per cent) had previously had a Dikkop, as compared to 6 (17·6 per cent) that had salted from horse-sickness fever. There is no doubt, therefore, that recovery from the severe form of Dikkop offers less protection than from the horse-sickness fever. It is generally expected from a virus that proves fatal for the greatest number of horses that recovery will produce an immunity that stands subsequent tests better than that observed after injection of a virus that is less fatal In this respect we have two good examples to compare, the ordinary virus (O) and the Tzaneen virus (Tz). Between these two extremes we can range a number of viruses that vary in virulency, and thus also in the immunity produced by recovery; such other viruses utilized by us were Bulawayo (B), several strains of Onderstepoort (O.P.), etc. The test of immunity obtained after recovery from the disease caused by one kind of virus can be satisfactorily carried out by injecting the immune animal with another virus. In this respect quite a number of experiments were made, of which a few will serve to illustrate better what is meant by different quality of immunity. In the year 1904–5 some horses that had recovered in horse-sickness experiments were exposed to natural infection. They had all been inoculated with only one strain of virus, viz. "O." One of these horses on the then Government Experimental Farm "Tzaneen" showed a breakdown, and blood was collected from it and used as virus (Tz). This virus having broken, "O" immunity was then considered to be stronger than "O," and tests to this effect were undertaken, in the hope of replacing later the weaker (O) virus by the stronger (Tz) virus for the purpose of immunization. A number of mules, totalling 139 all "O" immune, were then tested, with the result that 12 mules (8 per cent) showed horse-sickness fever, 4 Dikkop (3 per cent), and 1 died (0·7 per cent). Similarly a number of "O" immune horses (17) were injected, with the result that 5 (29 per cent) showed the horse-sickness fever, 3 showed symptoms of Dikkop (17 per cent), 1 died with symptoms of Dunkop (6 per cent), 1 of Dikkop (6 per cent). There were only 7 animals out of the 17 (41 per cent) that did not react; some of the tests had been made within a few months of recovery, and on horses that had been tested once, twice and three times with "O" virus and in large quantities. In the same year a virus was obtained from an "O" immune mule that recovered from a breakdown in Bulawayo (B virus). The "O" immunity of mules was also tested on 36 animals by inoculation with this virus, with the result that 7 (= 19 per cent) showed the horse-sickness fever, 6 (16 per cent) showed Dikkop and recovered, 1 had a doubtful reaction and 6 (16 per cent) died. Of 9 "O" immune horses tested with the same virus, 3 (33 per cent) showed Dikkop and 4 (45 per cent) died of horse-sickness, only 2 showed no reaction (22 per cent). There was thus ample evidence to show that "O" immunity can be broken to a considerable extent, and it remained to be seen to what extent immunity would hold that was produced by the recovery from either of the two breaking viruses. The immunization of equines was undertaken accordingly. In the subsequent tests it was found that of 59 "Tz" immune mules tested with "O" virus 47 (80 per cent) showed typical fever reactions, 2 (3 per cent) showed the symptoms of Dikkop and recovered, 5 (8 per cent) died of horse-sickness. Of 19 "Tz" immune horses tested with "O" virus 7 (38 per cent) showed fever reaction, 5 (26 per cent) showed Dikkop, 2 (10 per cent) died of horse-sickness and 5 (26 per cent) gave no reaction. Of the "B" immune mules tested with "O" virus all reacted and recovered, likewise did one horse. The immunity obtained by "Tz" virus was tested against "B" virus in 13 mules, when 8 (61 per cent) showed horse-sickness reaction and 1 (8 per cent) died, and in 4 horses, when 2 (50 per cent) showed horse-sickness fever reaction and the third one showed Dikkop. The "B" immunity was tested against "Tz" virus in 5 mules, when 3 (60 per cent) gave horse-sickness fever reaction, and in 2 horses, when 1 died. It is thus evident that we cannot speak very well of an immunity obtained from one strain to be better than that obtained by recovery from another. Mules and horses immune to any of the viruses will react again to some extent to another virus injected. There were a number of mules or horses that did not react to the injection and proved completely immune. Whilst thus breakdowns are on the one hand caused by a particular virus, immunity on the other hand is also connected with the animal itself. It has been observed that a given virus that has not the faculty to break an immunity may acquire it. It has been our experience that a virus of a given strain will not break the immunity produced by that strain, even when such large quantities are injected as required for hyperimmunization (10 litres) so long as the virus injected is of the same generation or passage; when of a higher passage, a difference of one or two may cause breakdowns. This has been observed in the "O" virus to take place after the 60th passage and in "Tz" virus after the 20th passage; such breakdowns are, however, by no means frequent; whilst such increase of virulency does not offer any difficulty to be understood, virulency of pathogenic micro-organisms being commonly increased by the passage through an animal, the reverse observation, viz. the strain of a lower generation of the same virus breaking the immunity obtained from recovery of a higher one, is an exceptional one. This has been noticed to take place with the "Tz" virus, which in several other respects shows peculiarities. Here it is not the change in virulency produced by a higher passage, but probably by the influence of the horse through which the virus passed. Under natural conditions some such process must be expected to take place; it is otherwise difficult to understand how there can exist so many different strains of horse-sickness breaking the various immunities.

Experience in practice has shown that horses that have had one breakdown in immunity (relapse, aanmaning) acquire a better immunity, and further breakdowns occur to a much lesser degree. This observation has some support by our own experimental results. Of 194 horses that as a result of one virus injection developed and recovered from a Dikkop reaction, 46 showed a second Dikkop reaction when tested with a different virus (23 per cent), of those that had had two Dikkops only one showed it to subsequent tests (2 per cent). We have seen this occurrence more than once, but three Dikkops are rarely found in the same horse. Although this figure perhaps does not accurately reflect the true state of affairs, nevertheless it may serve as an indication that the immunity for horse-sickness increases with a second breakdown. There are no horses on record with four Dikkop breakdowns, but some with four different reactions, some of which include two or three Dikkops. The statistics given above about the breakdown of immunity in the different years due to natural infection refer to any kind of immunity obtained from any virus, although the majority of horses had been injected with more than one virus; in most cases with two (Tz and O). Of 427 horses that had been injected with two virus (O and Tz) 122 had shown relapses (26·2 per cent) of which 40 died (9 per cent); 140 horses had been injected with three or more different viruses; of these 24 showed relapses (10·7 per cent) of which 9 died (6·4 per cent). There is accordingly no doubt that resistance to three or more viruses increased the immunity in a similar manner as recovery from breakdowns does, indicating in which way it may be improved artificially.

Therapeutic and Preventive Treatment of Horse-sickness.—As far as the treatment of sick horses is concerned no success has been obtained. It was undertaken on a fairly large scale, with the object of internal disinfection by means of chemicals and serum or by acting on the vasomotor system by means of drugs. Therapeutic interference on the heart's action would be called for in cases of Dikkop and has been tried, but experience has shown that the object is apparently best achieved in leaving the horse alone. Horses suffering from Dunkop frequently choke when drenching is attempted. Claims of successful treatment have been made both for horses and mules, but in view of our experience must be interpreted as coincidences, in particular in mules, that naturally recover more frequently than horses.

Preventive Treatment.—The object is to protect the animal against infection. There are two ways of doing this: (1) by preventing the animals from being bitten by the infection-carriers; (2) by rendering the animals immune. If the immunization process were a simple one, requiring only an injection of vaccine, as in the case of anthrax, etc., and if the immunity so conveyed would be an absolute one, then undoubtedly vaccination would be the ideal preventive treatment under all conditions. At present the process of immunization is still a cumbersome one by force of circumstances, and the immunity, as shown, is not a complete one and can never be expected to be so. As yet in the inoculation of mules a loss of 1-2 per cent and in the case of horses 3 per cent occurs, and deaths subsequent to breakdown in immunity from 3-5 per cent in the former and 5-10 per cent in the latter. The immunization for the proprietor remains mainly a commercial proposition in which the issue is whether it is more profitable to keep an immunized animal running free on the veld and to be of use at night time, risking breakdowns in immunity, or to lock it up in a stable during the night and only to use it during the day. It is from this point of view that the immunization of mules has been undertaken and is still carried out, the total number so inoculated since the introduction of the method in 1905 amounting to about 30,000. In the case of horses it has been attempted in an experimental way on a limited scale (about 4000) under the conditions of permanently infected areas.

When immunization is not undertaken, individuals or small numbers of equines can be efficiently protected by stabling, provided that the stable is mosquito proof ; or in the absence of such a stable, by smoking it out and maintaining the smoke during the night—horses and mules generally suffer but little from the smoke. In cases of individual horses to be used during the night (riding and cart horses) a dressing of the skin with some deterrent drugs is much made use of, commonly paraffin, alone or mixed with oil, by rubbing the animal down with a rag damped with it.

Where a greater number of horses is involved and the horses are not accustomed to handling and stabling, resort is taken to removing them to areas that are relatively or absolutely free from the disease ; in this case they are moved into higher situated farms, on top of hills or plateaux, or into districts known to be free. Where, however, the disease appears in areas under ordinary conditions free of it and surprises the farmer, then he is usually belated in his preventive measures and, as in the case of the breeders, has not sufficient stabling for the wild mares. A quick removal to higher altitudes or disease-free areas seems then to be the only resource left.

The dipping of horses as a protection has been suggested for some time, and has been carried out on several farms and been claimed to be successful, the dip that was used being the usual tick-destroying liquid containing arsenite of soda, with or without the addition of soft soap and paraffin, whilst some use an addition of linseed oil and paraffin ; dipping to be repeated weekly or even at shorter intervals. Indeed if a composition of a dip could be found that would deter the biting insects, then prevention of horse-sickness would become possible on all farms, although it would not be the ideal solution. Experiments undertaken to this end, and carried out under the conditions of a severe infection, show that satisfactory results may be expected if the intervals are sufficiently short, so that the deterrent material is constantly present on the horse. During the horse-sickness season 1917 an experiment in Onderstepoort was carried out that was intended to decide on principle the usefulness or otherwise of dipping. A mixture of possible deterrents was used, an arsenite of soda solution containing soft soap and aloes with a surface layer of paraffin oil, crude fish oil, cod liver oil, tar oil, fœtid animal oil, crude pyridin, and nitrobenzol ; in this way it was hoped to act on all the senses of a blood-sucker, the individual active ingredient to be worked out later. A total number of 20 horses were exposed, 10 acted as controls. Of the latter 6 died, viz. 60 per cent ; of the horses dipped at seven-day intervals, 2 contracted the disease of which 1 died, viz. 30 per cent, and of the horses dipped at three-day intervals 1 died of horse-sickness (10 per cent). Hopes are thus justified that by dipping, protection against horse-sickness can be obtained to a considerable extent. Experiments carried out on identical lines the subsequent year were frustrated entirely owing to the caustic effect of the fish oil then used, thus indicating a possible danger from the use of such ingredients.

The Immunization of Mules and Horses against Horse-sickness

Hyperimmunization.—Collection of Serum.— The recovery of an equine from horse-sickness does not give rise to an appreciable quantity of potent anti-serum, but such a serum can be obtained by hyperimmunization The practice adopted by us to hyperimmunize is simply to connect the jugular vein of the virus-producing horse with that of the serum horse, when a steady flow from the former into the latter occurs, at about the rate of 500 c.c. per minute with a cannula of 3 mm. and tubing of 1 cm. lumen. Experience has taught us that in order to obtain a potent serum a horse must be infused at least to the amount of 10 litres ; that the succeeding bleedings for serum, at the rate of 5-6 litres per time, must take place at four succeeding intervals of five days each. Fourteen days after the last bleeding of the first infusion the hyperimmunization is repeated for a second time and again for a third time at the conclusion of the second one. All the serums so collected are stored in a cool room at freezing-point and mixed with a carbolic acid solution, so that the bulk contains about 0·5 per cent carbolic acid. At the present time only one virus is used to hyperimmunize horses, viz. (O), the one that has proved to be the most virulent one. Hyperimmunization by infusion offers technically no difficulties, care being taken to prevent an influx of air by placing a glass tube between the rubber tubing of the infusing with the infused horse. The arrival of air bubbles due to insufficient fitting of cannula and connection can be controlled. Horses can stand a fair amount of air bubbles, provided they are not of great bulk. Generally the infused horse does not react at all to the infusion, and throughout the operation shows no deviation from a normal state of health. Sometimes during infusion the respiration becomes accelerated and superficial, then it is necessary to discontinue until these symptoms have disappeared, after which usually the operation can be finished. In some cases it is noted that a short arrest of the infusion does not prevent the respiratory trouble, then it is advisable to postpone the further transfusion to one or more hours later. In very rare instances it has been noted that horses cannot be hyperimmunized on account of these disturbances. Shock, viz. sudden death, is noted in about 1-2 per cent of cases infused, and may appear as soon as the inflow of blood begins, or any time during the process or at its conclusion; most frequently during the operation, rarely during the twenty-four hours following it. It shows itself as a sudden acute dyspnœa with violent movement of both chest and abdomen, open nostrils, staring eyes, and an acute cardiac action, indicated by a recurrent negative venous pulse, large waves succeeding each other rapidly along the whole length of the *vena jugularis*. The gums suddenly show paleness, the horse staggers and drops, and dies in a few seconds, showing spasms of the limbs and neck. The symptoms of shock are to some extent connected with the calibre of the cannula and the rate of transfusion, a large lumen and a quick rate, or sometimes the use of a fresh virus horse apparently increases the danger. Another set of symptoms occurs sometimes towards the end of the transfusion or shortly afterwards, viz. slight colic accompanied with increased respiration, slight perspiration, tremor in the muscles of the shoulder, the arm, and the thigh; frequent attempts at micturition, and finally evacuation of fæces, first of normal consistence and later pultaceous and watery. After this defæcation the horse improves rapidly and shows this usually by shaking itself several times, accompanied by snorting. More severely affected horses go down and take up a sternal position or lie flatly stretched out on the side; occasionally a horse may die. Death may occur within the twenty-four hours following the infusion. These phenomena are probably due to intoxication by the infused blood. Post-mortem reveals no abnormalities in the case of shock, not even hæmorrhages in the left endocardium. In the case where abdominal disturbances were noted the presence of acute congestion of the large intestines has been recorded. The infusion of such a large quantity of virus as contained in 10 litres of blood does not cause any reaction in the infused horse that might be interpreted as horse-sickness, provided the infused horse possesses an immunity to the same virus and the same passage. Fevers not due to horse-sickness may be noted when the virus horse has been a virus reservoir for Biliary fever or Pernicious Anæmia, which will then be transmitted. In both cases susceptible horses will develop the typical diseases. The occurrence of the former disease can be somewhat excluded, by taking care to use Biliary fever immune horses, but even here an occasional breakdown must be expected. Such horses have difficulty in recovering sufficiently to be used as serum horses, and Pernicious Anæmia horses become entirely useless. It is occasionally noted that the infusion of 10 litres of blood causes the appearance of an icterus in the first few days after the operation, which in the absence of Nuttallia equi must be considered to be caused by the breaking up of the corpuscles infused. This icterus may or may not be accompanied by fever, which begins as a rule with a high exacerbation and more frequently by a lytic than critic descent, and returns to normal within a few days.

Inoculation of Mules.—In the case of mules the same virus is used as that for the hyperimmunization; it is obtained from a horse previously injected with this virus. At the acme of the fever the horse is bled; the blood is defibrinated and mixed with an equal quantity of a solution consisting of water 500 c.c., glycerine 500 c.c., sodium oxalate 5 grammes, carbolic acid 5 grammes. The method employed is that called the "simultaneous" one, viz. serum and virus are injected at the time of the operation, the virus in all cases intrajugularly, the serum subcutaneously or intrajugularly. The quantity of serum varies from 300 to 350 c.c. for small and large animals. Lately the intrajugular injection has been recommended. As a result of this injection the mule will develop an attack of horse-sickness that corresponds in the majority of animals to the horse-sickness fever or to the Dikkop described, rarely to a distinct Dunkop. The latter usually ends with death; death may also occur from Dikkop.

Inoculation of Horses.—The serum employed is the same, the method is that called the "succedaneous" or "progressive" one; two viruses are employed; a first one to give the initial or ground immunity, and a second one to improve the immunity, and to bring it to the standard of that given to mules. The first virus is an attenuated one, that called Tzaneen (Tz); the second is a highly virulent one, that called Ordinary (Pretoria O). The first virus is injected alone (in the dose of 5 c.c.) into the jugular vein, the second virus is injected on the sixth day (in the dose of 3 c.c.) together with the serum, the latter at the rate of 0·75 c.c. per kilo live weight, and is repeated at the rate of 1 c.c. per kilo live weight on the eighth day. There are accordingly rather large quantities of serum required. This method is the outcome of a considerable number of experiments, in which the main facts ascertained were that it is impossible to immunize horses by simultaneous injection of virus and serum, because in the case of a strong virus (O) the mortality is too great, and in the case of a weak one (Tz) no reaction and then no immunity may result. The method is a careful balancing out of two viruses with a serum injected at intervals, that will ensure a reaction in all cases with a minimum of mortality and the maximum of immunity. This maximal immunity is brought about by superposing the reaction caused by the second stronger (O) virus to that caused by the previous attenuated (Tz) virus, both controlled by the same serum. Two successive reactions with two Dikkops are occasionally seen. It is thus an immunity to two viruses.

Technique of Immunization.—There are required a smaller syringe for the injection of virus (10 to 20 c.c.) and a large one (50 to 100 c.c.) for serum. Before use and between use they must be sterilized. For the injection of virus an ordinary cannula is required, which is inserted into the jugular vein, and the required quantity of virus is injected. In the case of serum it is advisable to use a trocar and cannula of small calibre (that destined for bleeding of sheep or goats), to insert it in its whole length, and only then to inject the serum. Short needles should not be used, they are moved about each time when the muzzle of the syringe is inserted; they injure the intima of the vein, or even perforate it, and so may lead to a thrombophlebitis. With the use of a larger-sized cannula symptoms of shock must be expected; they are apparently caused by the forcing in of the serum at a quick rate and higher pressure, both being greater than in the usual transfusion. Most horses and mules support the serum injection without any disturbance—some, however, are affected, showing definite symptoms: tremor of the whole body, stretching of the legs so that the animal is slightly raised, arching of back and stretching of neck, accelerated respiration and pulsation; or they begin with crouching, flexing of the hind legs, the hoofs moving dorsally and the animal walking for a few steps on the tip of the toes. These symptoms, however, do not last long, and can apparently be prevented to a great extent by observing strictly the technique described. However, some serums cause decidedly more disturbances than others even if injected with the greatest care.

Observations concerning the Selection of Virus.—Much care must be taken in the selection of the virus. Experience in this respect has taught us many severe lessons. We have in South Africa two diseases in which the recovered animal acts as a virus reservoir. Recovery from Biliary fever or Pernicious Anæmia leave the blood of the recovered animal infective. If blood of such horses is used as horse-sickness virus, then the disease may be transmitted to susceptible animals. In the case of *Nuttallia equi* the difficulty is easily overcome by using blood that is at least a month old; in the case of Pernicious Anæmia such virus remains active for at least one year, during which period the horse-sickness virus frequently may undergo modification in vitro, that makes it unsuitable for use. Passing such a virus through a dog and back into carefully selected horses whose blood has primarily been tested by inoculating into other horses with negative results is resorted to with advantage.

Observations concerning the Preparation of Serum.—The method adopted by us for the production of a serum probably offers all or at least most of the guarantees to obtain and maintain equal quality or standard. Usually a large number of horses are used, about 30 at a time in each of the three hyperimmunizations, so that a given serum as used in the practice of immunization is really composed of the serum of ninety horses, viz. thirty that have been immunized once, thirty that have gone twice, and thirty that have gone three times through the transfusion. The fresh blood is left over night at room temperature to facilitate coagulation, the second day the weights under the covers are dropped on the clots below, and coagula that are not detached are loosened. The bottles are then placed in the cool room (± freezing-point), and after cooling the serum is siphoned off in flasks, and subsequently the contents are mixed in one large receptacle and finally bottled. Before collecting, on the occasion of the first bleeding, the serum of a horse must be tested on its hæmolytic potency. As a result of hyperimmunization by the infusion of large quantities of infected blood into immune horses, the serums of the latter may acquire hæmolytic qualities; so-called Isolysines are formed. Such serums when injected into horses or mules usually give rise to a dissolution of the red corpuscles, thus causing an acute anæmia, hæmoglobinuria and icterus, ending in most cases with death. This acute disease, which as a rule is of a rapid evolution and short duration, is observed in the majority of cases either the first few days after the serum injection, or it arises in the course of the horse-sickness fever reaction, when it is usually less fatal and does not affect all horses injected. Experience has shown that after subcutaneous or intrajugular infusions of defibrinated blood a greater number of equines respond with hæmolysines than after infusion of the whole blood from animal to animal.

Testing of Serum for Hæmolysines.—Immune serums are tested on their hæmolytic effect by mixing 2 c.c. of serum with ½ c.c. of washed red corpuscles not diluted, and placing them into an incubator at 37° C. for two hours, when the dissolution of the red corpuscles is indicated by the red staining of the supernatant liquid. The test is read on the following day. The intensity of the staining is used as an arbitrary index, which in our practice conforms to the scale: "trace," "slight," "distinct" and "complete" hæmolysis. Experience has shown that, if the mixture contains a number of serums that show the index "slight," then hæmolysis in the injected horses can be expected. Hence in the selection of the serums for use, all such serums that tend towards the mark "distinct" are excluded. When, however, the greatest number of serums show no evidence of hæmolysis, the inclusion of one or a few showing "slight" hæmolysis does not endanger the use of the material for injection. Under all conditions the serums showing distinct dissolution are excluded. The total number of horses that through this cause become unfit for serum production averages about 10 per cent after the first hyperimmunization. For the test usually the blood of ten different normal horses is required.

Immune serum may be the carrier of the virus of Pernicious Anæmia. Experiments have proved, however, that by storing the serum for at least a year the pernicious anæmia virulency dies out, whilst the horse-sickness immunizing qualities do not suffer.

Sequels to Inoculation.—A certain amount of danger is connected with the inoculation of horses in the transmission of both Biliary fever and Pernicious Anæmia from one horse to another, particularly of the latter disease. In one such experience we saw the disease subsequently in 60 per cent of the injected horses, it having been transmitted by the syringe from two virus reservoirs. The greatest care must therefore be taken that during the operation no blood is transported from one horse to another by means of instruments. This object can be achieved by using two or more sets of syringes that are sterilized after the injection of each horse.

Horses that are undergoing immunization require attention; they cannot be used during this period, and must be kept under shelter and given preferably a diet containing green food. Experience has shown that it is advisable not to make use of salted horses for some weeks after recovery. Mules that go through the immunization require less attention and may even be turned out, and are usually fit for work again in three weeks; horses usually in four weeks, but preferably should not be used before the sixth week, particularly after recovery from Dikkop.

During the last four years a most peculiar experience has been noted in connection with inoculated horses, viz. the occurrence of acute or **Mad Staggers,** caused by an acute parenchymatous hepatitis, resembling the human acute Liver-atrophy. The disease is also in a much lesser degree noted amongst non-inoculated horses and in some cases with a previous history of horse-sickness, so that we are inclined to consider the passing through horse-sickness and subsequent early exposure and working as contributory factors, acting by association with a primary cause that is not yet clearly understood, but perhaps of vegetable origin. This conclusion has been arrived at as a result of negative transmission experiments and by analogy with Senecio poisoning, which produces symptoms somewhat akin to Staggers and lesions in the liver that differ only in degree from those found in "Mad Staggers." The name "Mad Staggers" (Malziekte of the Boers) is an appropriate one, in most cases, the horses so affected showing most violent symptoms and becoming entirely uncontrollable, having a persistent tendency of pushing in the one forward direction until they are exhausted; recoveries occur rarely and are probably not more than about 10-15 per cent.

Immunization does not as a rule interfere with the horses' spirits or capacity for work. In a few cases that recover from severe Dikkops, when a *restitutio in integrum* of the heart muscle apparently does not take place, the horse soon shows symptoms of fatigue and dyspnœa when used under saddle or in harness.

A. T.

CONTAGIOUS PUSTULAR STOMATITIS

Stomatitis Pustulosa Contagiosa

Contagious pustular stomatitis is an infectious exanthema characterized by fever and lesions chiefly affecting the mucous membrane of the mouth and lips, and generally running a benign course. It commonly affects horses, and in rare cases has been transmitted to cattle, sheep, pigs, and birds. Cases have also been observed affecting the hands and face of stablemen attending infected horses.

The causal agent of the condition is unknown, but infection is transmitted by saliva of affected animals, and is conveyed by contaminated food materials, water, litter, mangers, nose-bags and grooming utensils, particularly sponges.

Young horses appear to be more susceptible than adults. The condition, however, is very contagious, and when once introduced into the stable is likely to affect most of the animals there. Recovery from one attack leaves some degree of immunity, the duration of which is not clearly established. The disease has not infrequently been confused with horse-pox.

The *period of incubation* is from three to eight days, and the duration of the attack is from ten days to three weeks.

Symptoms.—The symptoms commence with a primary fever, the temperature rising to about 104° F. and somewhat fluctuating. The pulse is frequent, up to sixty, and full and large in character. The appetite is normal at first, but as the condition advances, prehension of food is painful and the animal refuses to eat unless provided with soft food. There is dribbling of saliva and smacking of the lips owing to the irritation, and the animal objects to the exploration of the mouth. The nasal mucous membrane and the conjunctiva are also injected. The mucous membrane of the mouth is hot and red, and small hard nodules up to the size of lentils can be felt on running the fingers over it, particularly on the inner side of the lips. Similar nodules are found under the tongue, on the gums, and on the inner surface of the cheeks. They may also extend to the skin and lips, the wings of the nostrils, and the nasal mucous membrane. When the latter is affected there is a slight muco-purulent nasal discharge which may dry on the margins of the nostrils. In rare cases lesions have also been seen in other parts of the body, such as the genital organs and the body surface. In three or four days these nodules become pustules, and in five or six days later they burst, forming ulcers with a red granulating base. In mild cases they heal fairly rapidly and leave no permanent marking, but in the more severe cases the ulcer penetrates more deeply and a permanent cicatrix remains. When the lesions are met with on the skin the discharge causes matting of the hair in tufts, and the formation of scabs under which healing proceeds.

The sub-maxillary and pharyngeal lymphatic glands are usually hot and swollen during the early stages of the disease, but gradually subside after the bursting of the pustules, while in very rare cases abscess formation has taken place in those glands.

Differential Diagnosis.—Contagious pustular stomatitis may be confused with horse-pox, coital exanthema, vesicular stomatitis, and with glanders.

In **horse-pox** lesions may be found on the mucous membranes as well as behind the pasterns. In stomatitis the main lesions are in the mouth, and there is an absence of lesions in the limbs.

In **coital exanthema** the lesions are principally met with on the genital mucous membranes, and especially after coition.

In **vesicular stomatitis** the lesions are essentially vesicles in the mouth and are not preceded by nodules nor followed by pustules.

In **glanders** ulceration of the nostril may occur, but mouth lesions are usually absent. The swelling of the lymphatic glands is persistent, and the condition usually runs a chronic course.

Treatment.—Isolate the animal as soon as possible, disinfect the place thoroughly, including mangers, watering-pails, sponges, bridles, and everything that would become soiled by saliva. Provide a soft diet, as of cut grass, steamed chaff, boiled oats, and mashes.

Medicinally only mild treatment is called for. Sulphate of magnesia ℥ii. twice daily in the drinking-water may be given, or similarly chlorate of potash ʒii. In the more severe and protracted cases quinine ʒss. and sulphate of iron ʒii. may be given daily in the form of a ball. An astringent mouth-wash may also be used, such as alum grs. v.-x. to ℥i. Lesions on the skin may be similarly treated with astringent disinfectants.

G. H. W.

VARIOLA; POX

The term Variola includes a series of contagious eruptive fevers which affect animals of various species with certain common manifestations. The eruption takes the form of a skin lesion, which is termed a "pock." In a typical case the pock runs through a series of five stages, to be described later.

Etiology.—Variola is a contagious disease, and is due to an ultra-microscopic organism which will pass through the porcelain filter. The virus is contained in the cutaneous lesions, but it is doubtful whether it is also contained in the blood, exhaled air, and the various excretions. There is some doubt as to whether the virus is the same in each of the species of animals affected, a common view being that the causal agent is the same, but somewhat modified by the transmission through different species of animals. On the other hand, some authorities are of opinion that the variolæ of the different species are quite independent. The bulk of experimental and clinical evidence, however, leads to the former view.

Lesions in Variola.—As already indicated, in a typical case of variola the lesions run through a series of five stages. *The roseola*, or first stage, consists of a diffuse redness of a small patch of the skin resembling a flea-bite, and due to hyperæmia. On this area the skin papillæ become enlarged, forming small hard elevations or papules. This constitutes the second or *papular stage*. From the congested vessels of the papules an exudation of lymph takes place between the corium and epidermis, thus producing a small vesicle and constituting the third or *vesicular stage*. In benign cases the vesicles remain discrete though multiple, but in severe affections they may become confluent. This is the critical stage of the affection. Later the serous liquid becomes turbid or purulent, thus converting the lesions into pustules and constituting the fourth or *pustular stage*. The pustules next give way to the formation of crusts or scabs which may be yellowish or blood-stained, and which become detached. This constitutes the *desquamative* or *crustaceous stage*.

In most of the variolæ there is an interval of about three days between the various stages, so that the full course in a typical instance is from fifteen to twenty days after the onset of symptoms. In benign cases of variola there is no permanent mark left, but in the severe forms, where the deeper layers of the skin have been involved, a permanent scar remains. This is known as the *pock mark*.

The variolæ of the domesticated animals are :

Variola Vaccinia (Cow-pox).
Variola Equina (Horse-pox).
Variola Ovina (Sheep-pox).
Variola Caprina (Goat-pox).
Variola Suilla (Swine-pox).
Variola Canina (Dog-pox).

Variola Vaccinia (Cow-pox)

This is a comparatively benign contagious disease, and if uncomplicated is never fatal. The systemic disturbances it induces are very slight. It is called "vaccinia" because it is most often seen in *cows*, but all bovines are susceptible to the condition. It is also transmissible to the horse, sheep, goat, and to the human being.

Jenner's view was that cow-pox arose from the inoculation of material exuding from the legs of horses affected with "grease," which he had failed to differentiate from horse-pox.

Cow-pox is usually introduced into a herd by means of a newly purchased cow, and is then spread amongst the herd by the hands of the milkers. It is also stated that men who have handled horses with horse-pox may infect cows. An outbreak in a herd may also be introduced by a vaccinated person.

The *period of incubation* in naturally occurring cases is four or six days, but if inoculated through a scratch or wound the period may be reduced to two or three days. In an outbreak a period of ten or twelve days generally elapses between the introduction of a diseased cow and a widespread infection of the herd.

Symptoms.—The lesions of vaccinia are chiefly local, occurring most commonly on the udder and teats. Occasionally they are met with on other parts of the body, such as the mouth, the vulva, and on the inner surface of the thighs. On rare occasions similar lesions have been met with on the scrotum of the bull. The first indication is usually a soreness of the teats and a tendency on the part of a previously docile cow to kick while being milked. On examining the parts the teats may be found swollen and small nodules felt about the size of a split pea. In a few days these become converted into vesicles with a diameter varying from that of a threepenny-piece to a shilling. They are circular on the udder but elliptical on the teats and depressed in their centre ; they contain clear transparent lymph. In the latter situation the vesicles are ruptured at an early stage by the milkers, and the scabby stage is developed quickly. On the udder, however, the vesicle develops into a yellow pustule, the scabby stage following several days later. The lesion is surrounded by a red zone. When the scab becomes detached the underlying skin is pink in colour and a slight scar is produced. This, however, has a tendency to disappear, so that permanent marking is very rare. The lesions in cow-pox are rarely very numerous, but reinfection may occur with the production of a series of lesions. There may thus be seen simultaneously several stages in the one animal. If no reinfection takes place, recovery may be complete in from fifteen to twenty days, but in cases of reinfection a case may be protracted. The veterinary surgeon is seldom called in till the first case has arrived at the vesicular stage, and by that time it has usually spread to other cows. Systemic symptoms are very slight. There may be slight fever with some reduction of the appetite and sometimes temporary cessation of rumination, while the milk yield may also be somewhat diminished.

Differential Diagnosis.—The lesions of cow-pox may be confused with :

(A) *Varicella* or *Water-Pock.*—In this condition the lesion consists simply of small vesicles, which are pointed and not indented on the surface, affecting the udder and teats. They contain a thin watery liquid and are very superficial, and do not run through the characteristic stages of cow-pox. The duration of the affection is from seven to ten days, unless there is a succession of lesions which may prolong the attack.

There is no systemic disturbance or surrounding red zone. A number of cows may be affected simultaneously.

(B) *Impetigo.*—A pustular eruption of the udder and teats. The lesions in this condition, of a diameter averaging about ½ inch, are yellow and flat on the surface. They are quite painless, and do not run through a series of stages as in the case of cow-pox. The affection is very benign, but has a tendency to spread to other cows by the milker's hands. It calls for very simple treatment on similar lines as cow-pox. It rarely lasts more than a week or ten days.

Treatment.—There is a natural tendency for the lesions to heal spontaneously, so that no drastic measures are required beyond keeping the lesions clean and applying some mild antiseptic lotion such as solutions of salicylic acid, boracic acid, or alum. Agents such as carbolic acid, which might taint the milk, should be avoided. After cleansing the lesions a little dry boracic acid should be dusted over them. Where possible, the affected animals should be separated from the rest of the herd and should be milked by separate attendants. If that cannot be done the healthy cows should be milked before the affected ones.

Internal treatment is rarely necessary, but in cases associated with fever small doses of sulphate of magnesia ℥ii.-iv. may be administered in the drinking-water.

Vaccination.—Vaccination is the taking of vesicular material from an animal affected with cow-pox and inoculating it into an animal of another species with a view to protecting that animal from its own form of variola.

For vaccination in the human subject the vaccine is not taken from cows' teats and udders in naturally occurring cases, but from artificially produced cases in young calves. For the preparation of the vaccine strong healthy calves are taken, and after being proved free of tuberculosis by testing, their abdominal wall is shaved and thoroughly cleansed with ether. The skin is then scarified and inoculated with lymph taken from a naturally occurring case of cow-pox, or from a previously inoculated calf. In five or six days the vesicular lesions are developed on bright red bases, and from them the lymph is collected by means of a spoon or special forceps. It is then mixed with either glycerine or a little carbolic acid and preserved in sealed glass tubes. The calves are allowed to recover and are afterwards slaughtered and a careful post-mortem examination made. If the calf proves to be perfectly healthy the lymph is passed into stock for use. If, on the other hand, the calf shows any trace of disease the lymph is destroyed. The calves treated in this way are, after recovery, quite fit for human food.

When this so-called calf lymph is inoculated into a human being it gives rise to a lesion of variola vaccinia, and it has been abundantly proved that a human subject who has had vaccinia is less likely to contract smallpox than an unvaccinated person, and if he should contract it the disease runs a much milder course. This was first observed and proved by the fact that milkers who had been affected with vaccinia were generally immune to smallpox, and so escaped infection during severe outbreaks of that disease.

One strong objection that was raised to vaccination was the supposition that smallpox and cow-pox were two absolutely different diseases. Although cow-pox could be transmitted to man, it produced lesions resembling those occurring in cattle, and not very similar to those of smallpox, except in the course of stages through which they ran. On the other hand, smallpox could not be inoculated directly into bovines. It has, however, been shown that smallpox can be inoculated indirectly into bovines by modifying the virus by passing through monkeys. The lesions thus produced in cattle are almost identical with those of variola vaccinia. Thus a close relationship, even if not identity, of smallpox and cow-pox has been established, and it is only reasonable to expect that recovery from an attack of one should confer a certain amount of immunity against the other. Other objections to vaccination are chiefly connected with the alleged possible introduction of other diseases along with the vaccine, such as tuberculosis, syphilis, etc. The proper preparation of calf lymph, however, prevents the possibility of setting up tuberculosis, while syphilis is unknown in bovines. In those cases where the latter disease has been set up it has been the result of the very objectionable practice of inoculation of one human being from another. Septic organisms are sometimes inoculated along with the vaccine as the result of dirty procedure. The conclusion then is that vaccination, if properly carried out with pure calf lymph, cannot produce any serious ill-effects whatever, but, on the other hand, it may and does produce a strong degree of immunity against one of the most dreaded of human diseases.

Variola Equina (Horse-pox)

Horse-pox is a benign form of variola, but rather more severe than cow-pox, and is much less commonly met with. Horses of all ages and both sexes are susceptible to the disease. By many authorities it is regarded as identical with cow-pox, and certainly variola is intertransmissible between the two species.

When introduced into a stable it spreads fairly rapidly from affected to healthy animals by means of grooming utensils, the hands of attendants, bandages, contaminated litter, etc. It may also be spread by the shoeing smith and on rare occasions by coition. Horse-pox has also been transmitted to man.

The *period of incubation* is from five to seven days, and the disease runs its course in an uncomplicated case in eighteen to twenty days after the onset of symptoms. If neglected, however, the course may be prolonged.

Symptoms.—The systemic symptoms in horse-pox are not often very marked. There may be a preliminary rise of temperature 2° or 3° with injected mucous membranes and pulse increasing in volume and frequency. The horse may be somewhat off its food, and falls away in condition. In other cases the systemic symptoms are so slight as to pass entirely unnoticed. Lesions then develop on the skin and certain mucous membranes. The skin lesions in horse-pox are usually seen in the hollow of the pasterns and on the back of the limbs, but may be observed in other parts of the body. The skin becomes swollen and tender, and on it the characteristic lesions of variola are developed. Nodules (papular stage) may be detected and pass through the vesicular, pustular, and scabby stages successively. In this situation, however, the pustules are not easily recognized, and exudation often takes place on the surface, causing a matting of the hair. In this condition the true nature of the disease may readily be overlooked and be mistaken for grease, thus allowing the disease to spread throughout the stable. This discharging stage generally occurs about the ninth or tenth day after infection, and continues for four or five days. The lesions may also be found affecting the mucous membrane of the mouth, nose, conjunctiva, and sometimes the genital mucous membrane. When the mouth is affected the eruptions may be seen inside the lips and cheeks, on the under surface of the tongue, and on the gums. Rarely they extend up the face from the angles of the mouth. These lesions commence with the formation of nodules and pass into vesicles with straw-coloured contents, and then develop into pustules. They then burst, leaving a deep red depression. As in the case of most lesions of the mouth they heal rapidly. The affection of the mouth causes frequent smacking of the lips and dribbling of saliva, and may interfere with feeding. When lesions are present in the nostrils they give rise to a yellowish nasal discharge. When the eyes are involved there is an overflow of tears and often some opacity of the cornea. When the genital organs are affected the eruptions may be seen on the penis of the male, which may become swollen and pendulous. In the mare the vulva also carries eruptions and the lips of the vulva are swollen. The lesions have a diameter varying up to ½ inch, and may occur in successive crops, so that several stages may be observed at the same time.

Differential Diagnosis.—Horse-pox must be differentiated from **grease, contagious pustular stomatitis,** and **contagious acne.**

Grease will be distinguished by the chronic nature of the affection, with its extremely offensive odour, and by the absence of lesions in the mouth and genital organs. Moreover, there is no tendency for grease to spread throughout the stable.

Contagious pustular stomatitis may be distinguished by the fact that the lesions are essentially pustular and do not pass through the stages of variola. They are met with under the mucous membrane of the mouth, and are easily seen inside the lips. On passing the finger along the inner side of the lips the lesions feel like small shot under the mucous membrane, and when they burst they look as though a small piece has been clean punched out. It is very contagious, and usually spreads throughout the stable. The course is run through in about twelve days. In this affection there is an absence of lesions about the limbs.

Contagious acne is perhaps less likely to be confused with horse-pox in that the lesions mainly affect the skin of the back and withers, and are rarely found on the mucous membranes and the heels. It causes very little systemic disturbance.

Treatment.—Affected animals should be isolated at once, and should be attended to by separate grooms. On account of the lesions of the mouth a laxative and soft diet must be given, such as bran mashes, linseed mashes, boiled oats, boiled roots, and steamed hay. Mild febrifuges such as potassium nitrate and sulphate of magnesia may be administered in the drinking-water The lesions on the mucous membranes may be dressed with a weak antiseptic lotion, such as boracic acid and alum lotion āā gr. v.-x. to ℥i. The skin lesions should be dressed with an astringent and disinfectant solution, such as tincture of iodine 1-10, or copper sulphate, or zinc sulphate, gr. xv. to ℥i.

A thorough disinfection of the premises must be carried out, paying special attention to the manger, watering-pail, harness, grooming utensils, and litter, all of which may be the means of conveying the disease to healthy horses.

Variola Ovina (Sheep-pox)

Sheep-pox is the most virulent of the variolæ of the domesticated animals. It is very highly contagious, and may be spread by mediate or immediate contagion. As the name indicates, it mainly affects sheep, but it may also affect goats, and it has been transmitted to the ox, horse, and man.

Two forms of the condition are recognized: (1) a *benign* or discrete, and (2) a *malignant* or confluent sheep-pox. In the former the vesicles remain separate from each other and the mortality is comparatively low, viz. from 5 to 7 per cent, while in the malignant form the lesions frequently coalesce and the mortality may be up to 80 per cent.

Infection may take place either by respiration, by ingestion, or by inoculation.

The virus appears to be very resistant to external conditions, and consequently may be conveyed by numerous contaminated media, such as the fleece, sheep's skin, fodder, and litter. An animal that has actually recovered may still convey infected material in his fleece for some weeks after, and therefore remain infective. Stock yards, markets and fairs, and railway trucks may also remain infective for several months after they have been occupied by infected sheep.

The period of incubation is from two to seven days in the virulent form, and from ten to twenty days in the benign. Young animals are more susceptible than old ones, and the incubation period is usually shorter in the hot weather than in cold.

Symptoms.—The first symptom is usually an acute rise of temperature several days before the development of the local lesion. The fever is very depressant and debilitating. There is loss of appetite, suspension of rumination, and an accelerated pulse and blowing respiration. A discharge escapes from the mouth and nose, and a cough may develop. Affected sheep tend to isolate themselves from the rest of the flock. The lesions may next be observed, and are best seen in those places where the wool is scarce, such as inside the forearms, the inner sides of the thighs, under the tail, and often about the face. They run through the characteristic five stages of variola, and commence with small red spots in the various parts of the skin, on which small elevations or papules develop in the course of a day or so. About three days later the papules have developed into vesicles which are usually flat on the surface and white in colour. In very virulent cases there may be hæmorrhages into the vesicles. The vesicles are followed a few days later by the development of pustules and increased swelling of the skin. Vesicles and pustules may remain discrete, or may be so numerous as to become confluent. The pustules are followed by the development of scabs which become dry and may be hæmorrhagic and fall off in the form of scales. In very severe cases the lesions may become gangrenous, while the sheep become very weak and prostrate. Eruptions may also occur with similar manifestations in the mouth, causing dribbling; in the pharynx and bronchi, causing coughing; in the eye, causing conjunctival discharge; and sometimes in the alimentary tract, setting up diarrhœa. As in the case of other forms of variola, the vesicles do not all appear at one time, and may be seen in successive crops. In an uncomplicated benign case the course of the affection lasts from three to four weeks. In the confluent and more severe outbreaks death is likely to occur during the third or vesicular stage.

Recovery from one attack confers a life-long immunity.

Differential Diagnosis.—Sheep-pox has been confused with **ecthyma**—a pustular eruption of the skin. In this condition, however, the affection is essentially pustular with pointed lesions, but it is not associated with any fever or systemic disturbance. Moreover, there is no evidence of contagion, and the condition is comparatively mild, and never associated with fatal results.

Treatment.—In the British Isles sheep-pox is scheduled under the "Contagious Diseases of Animals Act," and curative treatment is not permitted. In fact, whenever this disease is newly introduced into a district it usually takes a virulent form, and it is improbable that any curative measures would be justified by results. The better plan then is to destroy all affected animals and those that have been in contact. In benign outbreaks, however, in countries where treatment is permitted the sheep should be placed in shelter with a plentiful supply of fresh air, but not overcrowded, particularly in hot weather. Attention should be paid to cleanliness of the floor and litter. A generous diet of soft foods should be provided, such as the various corn or cake meals, together with pulped roots. Medicinally, a mild febrifuge should be given in the drinking-water, such as sulphate of magnesia ʒss.-℥i. and potassium nitrate ℥i.; small doses of quinine may also be prescribed. The lesions may be dressed with mild antiseptics, such as lotions of boracic acid, alum, or hyposulphite of soda.

Prevention.—Preventive measures consist in the prohibition of the importation of sheep from infected countries. If the disease has broken out slaughter of all affected animals is best carried out immediately, with strictest disinfection of the premises, and the destruction of all infective material. If any of the sheep are allowed to remain a quarantine should be established, prohibiting the removal of sheep to or from the premises for at least three months, and preventive inoculation may be practised in such cases.

Ovination or Preventive Inoculation for Sheep pox.—This consists of the inoculation of sheep with material obtained from the vesicular stage of a mild case of sheep-pox. The inoculation is made under the tail or the inside of the ear, or the thigh, by means of a hollow needle or a lancet. It may also be carried out by a hypodermic syringe, or by way of a thread soaked with the virus inserted by means of a suture needle. In a typical case a lesion develops at the seat of inoculation and results in a mild attack of sheep-pox terminating in recovery, and leaving a strong degree of immunity which may last for several years. Occasionally, however, a virulent attack of sheep-pox results, which may terminate with disastrous results. This is particularly the case if the procedure is carried out amongst lambs. When carried out in England the results were not very favourable, but at the present time in those countries where sheep-pox is prevalent excellent results are claimed for it, and it is stated that mortality amongst flocks that have been inoculated has been reduced approximately to 1 per cent by this means.

Variola Caprina (Goat-pox)

It is doubtful if there is an independent variola affecting goats, but the goat is susceptible to sheep-pox and to cow-pox, both of which may be contracted under natural conditions.

When *sheep-pox* affects the goat the disease usually runs a milder course than in the case of sheep, the vesicles being smaller and not so pronounced. In the case of goats affected with *cow-pox* the lesions are much larger than in the former condition, the vesicles being umbilicated.

In goats the lesions are generally met with in those parts of the skin where the hair is scarce and the skin thin, such as the inside of the thighs and armpits, the face, and the udder. On healing there may be a permanent scar. In young animals mouth lesions may also occur. In goat-pox there is also a slight fever and catarrh, but the effects are not very serious. The condition, however, calls for an urgent control when an outbreak occurs, owing to the danger of contagion to the other species.

Treatment.—Treatment is the same as for the other forms of variola.

Variola Suilla ; Variola Porcina (Swine-pox)

Swine-pox is a somewhat rare disease, and its origin is in doubt. It is said to have been contracted from the human being by means of contaminated litter, and it has also been spread from swine to goats, sheep, and calves. It is a very contagious disease, and its effects are more serious in young swine than in adults. One attack is said to confer prolonged immunity.

Symptoms.—There is a primary stage of fever, with elevated temperature, general listlessness, and loss of appetite, and from five to seven days after infection red spots appear on the skin and various parts of the body, particularly under the abdomen, inside the arms and thighs, on the head, and behind the ears. On these red spots papules are developed which in turn pass on to vesicles. These are followed by pustules and later scabs. As in a typical variola, there is usually an interval of three days between the stages. In the majority of cases the lesions remain discrete, but in severe cases they become confluent.

It is probable that some cases of Swine Erysipelas have been mistaken for swine-pox. Variola is more serious in swine than in goats or cattle, and is fatal in a considerable number of cases. Its importance is increased owing to the probability of its origin having been the human being or sheep, as in either case there is a great probability of the spread of the disease again to those subjects.

Treatment.—Preventive measures are of great importance for reasons already indicated, and in the case of an outbreak amongst swine probably the best course is to slaughter all affected animals and in-contacts. If treatment is adopted it should be on the same lines as for sheep-pox.

Variola Canina (Dog-pox)

It is doubtful whether there is a form of variola affecting the dog, but in many cases of distemper there are skin lesions developed, particularly along the inside of the thighs, the flanks, the axilla, and under the belly, which present characters closely resembling some of the lesions of variola. Many continental veterinary surgeons are of opinion that a true variola of the dog does occur and that puppies are much more susceptible than adults. Most of the cases recorded, however, correspond very closely with the cutaneous form of distemper.

Based on the assumption that canine distemper is a form of variola, a system of true vaccination with calf lymph as a preventive of distemper is often practised. The efficacy of the inoculation, however, is doubtful, but it is usually safe and quite harmless.

G. H. W.

COITAL EXANTHEMA

Vesicular Exanthema of the Genitals

This is a contagious disease met with in horses, cattle, and occasionally sheep, and more rarely in other species, transmitted during coition and characterized by vesicular and pustular lesions of the external genital organs. There is some doubt as to whether the affection in the horse is the same as that in cattle, but the affection of either one may be experimentally inoculated into the other. Although there are some points of resemblance of this affection to variola it is improbable that it has any association with it. One important point supporting that view is the fact that recovery from one attack does not confer any immunity since repeated attacks have frequently been observed in both horses and cattle.

The virus is at present unknown, but is contained in the lesions in the affected areas and in the discharges. Although the affection is generally conveyed by copulation, it may also be transmitted by other media, such as grooming utensils, particularly rubbers and sponges.

The *period of incubation* for both horses and cattle is from one to six days—rarely ten days—while the course usually extends from two to three weeks and terminates in complete recovery.

Coital Exanthema in the Horse.—*Symptoms.*—In the **stallion** the penis and sheath become somewhat swollen and congested, and on them are seen small vesicles which develop into pustules. The eruptions may extend to the scrotum and to the inner side of the thighs, and under the abdomen to the fore limbs. The glans penis may become swollen and the penis pendulous, and may result in paraphimosis. The free extremity of the urethra becomes involved and a muco-purulent discharge escapes, and there are frequent attempts at urination, which is often painful and difficult.

In the **mare** the lips of the vulva and the vagina are affected with the formation of vesicles and pustules arising on a congested mucous membrane, causing frequent urination with erection of the clitoris and a muco-purulent discharge which frequently cakes around the margin of the vulva. The mare appears to be constantly in œstrum.

In both sexes there is considerable irritation and resentment at handling the affected parts. The lymphatic glands in the groin may be a little swollen and œdema may extend forward under the abdomen. There may be some loss of appetite and a tendency to constipation, but except in severe cases there is very little elevation of temperature. In mild cases recovery occurs in a few days, while in severe cases the course may extend over a month or more.

Treatment.—Preventive measures consist in isolation of any affected animals, and the temporary withdrawal from the stud of all animals with sores on the genital organs.

Curative measures consist in giving a mild laxative internally, followed by a combination of sedatives and antiseptics to the urinary tract, such as hexamine, the salicylates, hyoscyamus, buchu and copaiba. Locally the affected areas should be kept clean with mild antiseptics and astringents such as collargol 1 per cent to 5 per cent, sulpho-carbolate of zinc, sulphate of zinc, alum, acetate of lead gr. v.-x. to ℥i. in each case, chinosol gr. i. to ℥i. or carbolic acid 2½ per cent. If paraphimosis occurs the penis should be suspended by means of a truss.

Coital Exanthema in Cattle ("Bull-burn"; "Fired").—This affection is much more common than the corresponding disease in the horse.

Symptoms.—In the **bull** vesicles and ulcers develop on the glans penis (*balanitis*), the sheath and the scrotum. There is a urethral discharge of a muco-purulent character and a similar discharge from the prepuce or sheath. The latter may not be very copious, but causes a matting of the hairs over the prepuce. There is frequent micturition and the animal stands with tail raised. The sheath and penis may both be swollen, causing phimosis or inability to protrude the penis. When the penis is examined it is found to be affected with red granular angry-looking spots, and in exceptionally bad cases there may be gangrene of parts of that organ.

In the **cow** the condition commences with a redness and swelling of the lips of the vulva and the vaginal mucous membrane, together with a mucous glairy discharge. On examining the mucous membrane, red spots may be seen which develop into vesicles, to be followed by pustules and ulcers with a diameter about that of a pea. The discharge when copious runs down the perineum and the thigh and may cause crusts painful to the touch. There is usually frequent and painful micturition with erection of the clitoris, as though in continuous œstrum. Affected animals frequently lash their tail about or stand with it partially extended, and in milch cows lactation is diminished.

In both sexes there is slight interference with appetite, while rumination may be partially suspended, and animals fall off in condition. There is a slight tendency to constipation, although occasionally diarrhœa may be met with. Occasionally there is some elevation of temperature, 1° or 2°, but it is rarely excessive. In ordinary cases the duration of the affection is from ten days to a fortnight, but it may last a month; recovery from one attack does not confer any immunity.

Treatment.—Affected animals should be isolated, though there is very little evidence that the disease is conveyed in any other way than coition. A laxative and nutritious diet should be provided. A mild laxative should be given internally, while locally an astringent and disinfectant should be applied. Simple white lotion (zinc sulphate and lead acetate āā gr. vi.-viii. to ℥i.) may be applied two or three times a day, or similar lotions as recommended for the horse. The use of antiseptic bougies made up with cacao butter and either perchloride of mercury, chinosol, lysol, or iodoform may be inserted into the sheath or vagina respectively.

As a preventive measure careful examination should be made of any newly introduced animal before admission into the herd, and thorough disinfection of the bull should be practised after service of a cow from another herd. The disinfection should include the penis and sheath and the nose and breast.

Contagious Granular Vaginitis of the Cow.—This is a contagious disease which is thought by some to be the same condition as coital exanthema. It is spread during coition and by the hands of the cowman in examining for it. According to some continental authorities, it is regarded as one of the causes of sterility in cows.

Symptoms.—These are not so exaggerated as in bull-burn, and there may be little or no systemic disturbance. Attention is first drawn to it by a slight glairy vaginal discharge. On examining the mucous membrane of the vulva stripes of congestion may be observed in the folds on the floor of the vulva and vagina ; the clitoris may also be congested. Small nodules or granules can be felt in or under the mucous membrane, especially along the course of the congested stripes. They increase in size and ultimately burst, setting up a slight mucopurulent discharge. There is frequent micturition and erection of the clitoris, with nymphomania and constant returning to the bull. The constant irritation of the lower part of the genital tract is said to cause the development of cysts in the ovaries, which may be felt on examination per rectum. A number of cows may be affected at the same time.

In the bull the affection is not so severe as in cows. The penis, however, may be red and similar small granules may be felt under the mucous membrane as in the cow.

Treatment.—The external genitals should be irrigated with mild astringent antiseptics as in the case of bull-burn, and followed by the insertion of similar antiseptic bougies. If cysts of the ovary can be felt they may be ruptured by manipulation through the vaginal or rectal wall, and this procedure is often said to promote fertility.

In connection with this affection it may be noted that on passing the fingers over the vaginal mucous membrane and the floor of the vulva in the healthy cow, small elevations may be felt consisting of the small glands of the mucous membrane. It is quite conceivable that these structures have been wrongly regarded as a pathological condition in a number of animals alleged to be affected with the above condition, especially when there has been a complete absence of any systemic affection. This is the view particularly held by those authorities who doubt the existence of contagious granular vaginitis as distinct from coital exanthema of cows.

G. H. W.

CONTAGIOUS ABORTION OF CATTLE

The losses in dairy herds resulting from contagious or epizoötic abortion are second probably only to those occasioned by tuberculosis in this and other countries where the maintenance of dairy herds forms a very large part of the agricultural programme. It is not only the loss of the calf which results from the disease, but a very serious fall in the milk yield, since in very many cases the abortions occur in the earlier stages of pregnancy.

Moreover, cows which have aborted may become sterile for at any rate a time, while it occasionally happens that a cow aborts twice or even three times in succession.

All abortions, however, are not due to an infectious disease. Injuries, bad food, purgatives, acute febrile conditions, exposure, excitement, uterine tuberculosis, and other causes may produce abortion. The usual course of the disease in a herd is that it follows the introduction of a new cow which may slip her calf or even carry it until full term and yet be a source of infection (Holth). Probably only one or two abortions occur during the first year, but the number increases enormously during the second year, and attains its maximum during the third. Calves which are born sufficiently near their full term are usually weakly, and easily succumb to some subsequent infection. Contagious abortion is really a specific catarrhal metritis, the abortion being only a symptom of the disease.

Bacteriology. — The causal agent was first described by Bang in 1897. *Bacillus abortus* is essentially a tissue parasite the predilection seat of which is the gravid uterus, in which it is found in large numbers, either free or contained in cells in the uterine excretion of aborting cows (Bang). It is a small non-motile and non-sporing bacillus, varying in size from that of the " fowl cholera " bacillus to that of the tubercle bacillus, and was classed by Nowak with the " hæmorrhagic septicæmia " group of organisms. In the lesions and in artificial media it frequently occurs in clumps, and in the latter it is often polymorphic. It stains fairly readily with the basic aniline dyes, but not by Gram's method. The larger bacilli frequently show granular staining, and resemble a number of small cocci. Primary cultures are somewhat difficult to obtain. Bang recommended for this purpose a medium consisting of gelatine agar to which 10 per cent of ox serum was added.

On account of the peculiar oxygen requirements of the organism when freshly isolated, stab or shake cultures are employed and growth is visible in from 48 to 72 hours as a zone a little (10-20 microns) below the surface. After frequent sub-cultivation it grows under ordinary aerobic conditions upon the surface of most media. The rapidity with which this change takes place and the luxuriance of the surface growth vary very considerably with different strains.

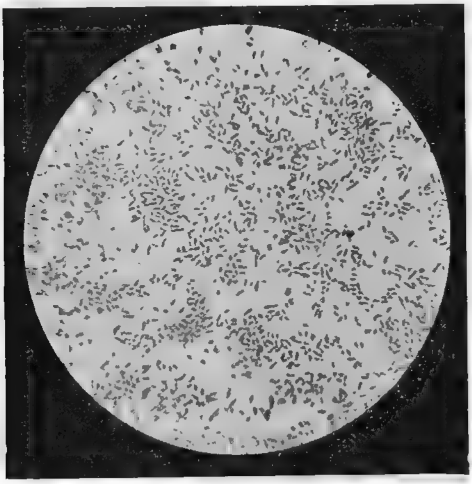

Fig. 11.—Bacillus of abortion, from pure culture. (Mag. 1000 times.)

Infection.—Natural infection is practically confined to cattle, and occurs in the vast majority of cases by the mouth as a result of the ingestion of fodder which has become contaminated. It is possible that in a few cases infection may be per vaginam through the agency of the bull, which, however, shows no symptoms of infection. Surface has described an epizoötic among guinea-pigs which was produced by Bang's bacillus.

Experimentally the disease can be conveyed to the mare, ewe, goat, and rabbit by ingestion or by parenteral injection. The subcutaneous injection of aerobic strains produced abortion in a cow fifty-two days after injection, and the organisms recovered from the fœtus were still capable of growing under aerobic conditions, showing that they are able to pass through the placental filter (Surface).

Vitality and virulence are maintained for very considerable periods. Uterine exudate which has been kept in an ice-chest for seven months has been found to be active, and a mummified fœtus nine months old was still infective. In artificial media the organism remains virulent for several years. In infected cows abortion may occur at any time after the third month of pregnancy. As a general rule the earlier in the pregnancy that infection takes place, the longer the abortion is delayed. When infection occurs late in the pregnancy the animal may abort within a few weeks.

Symptoms.—There are no definite premonitory symptoms, but there may be the usual signs of approaching labour. In cows which are in milk there is some falling-off of the secretion, and the milk may resemble colostrum. There is an opaque vaginal discharge which may appear a short time before the actual abortion, which usually occurs between the third and seventh months. If abortion takes place towards the end of the period of gestation a living calf may be born, but such animals are usually weakly and frequently die. The membranes are often retained by cows which have aborted, and such animals may show abnormally frequent œstrum, difficulty in conception, and may become sterile.

Lesions.—The typical lesion, which is a specific uterine catarrh, is best seen in animals which are about to abort. Between the uterus and the chorion there is a thick yellowish, odourless exudate of a glairy nature, and there may be a gelatinous œdema of the fœtal membranes which gives them a characteristic appearance after abortion. The cotyledons often appear as though macerated, opaque, and of a dull greyish colour.

The causal organism may be recovered in pure cultures from the exudate and membranes before or immediately after abortion, and from the liver, heart-blood, and alimentary canal of the aborted fœtus and the fœtal cotyledons.

Diagnosis.—The existence of the disease in a herd is recognized by the fact that a number of animals abort and at different times. The discharge is diagnostic, owing to its glairy nature and yellowish or chocolate colour. The chorion is thickened and œdematous, and the cotyledons are yellowish-grey in colour and softened. It is frequently possible to demonstrate the presence of *B. abortus* by the microscopic examination of smears from the placental exudate and fœtal liver or intestines, but the apparent absence of the specific organisms does not necessarily indicate that it was not a case of epizoötic abortion. The inoculation of suitable culture media may show the characteristic form of growth.

The Agglutination Test.—The serum of infected animals agglutinates suspensions of *B. abortus* in dilutions from 1 in 50 to 1 in 1000 or even more, while that of non-infected animals rarely agglutinates in dilutions of 1 in 50. In place of serum, milk (whey) may be used, but the results are not so definite.

Complement Fixation Test.—This test is a delicate method of diagnosis. While the serum of non-infected animals fails to fix the complement, that of infected animals shows a titre of from ·1 to ·002 or even less.

A positive reaction to either the agglutination or complement fixation tests does not necessarily indicate that a cow will abort, but it does show that the animal either is or has been infected with *B. abortus*.

Treatment.—*Curative.*—Medicinal treatment is useless.

Prevention.—Newly-purchased animals should be strictly isolated until they have calved, and cows should not be sent away to a bull. Cows which show any suspicious symptoms should be isolated and any discharge disinfected. The bedding should be frequently removed and destroyed. When an animal has aborted the fœtus and membranes must be burnt or buried in quicklime; all discharges and contaminated litter must be frequently removed and disinfected.

The uterus should be irrigated twice daily with disinfectant solutions, and any soiled parts of the body should be sponged. The floor and walls of the building must be thoroughly and frequently disinfected. A bull which has served infected cows should be cast, the prepuce irrigated, and penis washed with a non-irritant antiseptic and the long hair removed. In fairness to the rest of the community, animals which are known to be infected should not be disposed of, unless the fact is made known to the purchaser.

Protective Inoculation.—A certain amount of active immunity is acquired by infected animals, but it is probably a very slow process. Many animals abort only once, but it is by no means uncommon for cows to abort twice or even three times.

The Veterinary Department of the Board of Agriculture supplies a vaccine for protective inoculation. " Anti-Abortion A " is a saline suspension of living organisms obtained from the luxuriant surface growth on a special medium. It is injected subcutaneously into non-pregnant animals, which are not put to the bull until two months after the inoculation.

" Anti-Abortion B," which is used in the case of pregnant animals, is a similar suspension to the above which has been sterilized by heating for half an hour at 65° C. The dose employed is equivalent to half that of " Anti-Abortion A," and is repeated at monthly intervals up to the sixth month of pregnancy.

The results obtained with the living vaccine have been more satisfactory than those following the injection of the killed cultures.

J. B. B.

EPIZOÖTIC ABORTION OF SHEEP

It has been definitely established by the Departmental Committee on Epizoötic Abortion that the disease in sheep is specific. The disease in the sheep appears to be confined to definite areas in contrast to the bovine disease. This, as was pointed out by the Committee, is probably due to the fact that flocks are maintained in a more isolated manner than are herds of cattle, and there is consequently less movement of infected animals, and there is therefore less tendency for the disease to spread.

Although sheep may be infected experimentally with the bacillus of cattle abortion the naturally occurring disease was never found to be due to this organism, but to a specific vibrio. Abortion in sheep, owing to the fixed breeding period, is of a seasonal nature, and is not continually occurring, as is the case in the bovine disease.

A few naturally occurring outbreaks of vibrionic abortion have been observed in cows.

Bacteriology.—As it occurs in the lesions and discharges the causal organism appears as an " S " shaped body of from 1·5 to 3 microns in length and about ·25 of a micron in breadth at its centre, the ends being somewhat tapered.

The organisms may occur singly or in the form of spirals composed of several elements. They are actively motile, and multiply by transverse division.

They stain well with the basic aniline dyes, but not by Gram's method. One end of the organism occasionally shows a more intensely stained area, and granular staining is sometimes observed. The organisms appear to break up into small round granules which stain readily with toluidin blue. Cultures of the vibrio in artificial media prepared in the same way as in the case of *B. abortus* show similar peculiar oxygen requirements. After three days' incubation at 37° C. of shake cultures in agar-gelatin-serum a growth appears about $\frac{1}{8}$ to $\frac{1}{4}$ inch below the surface, which very closely resembles that of *B. abortus*. Growth at room temperatures is slow (ten days or more). The organism may also be cultivated in agar, gelatin or peptone broth; the gelatin is not liquefied. Growth does not occur on the surface of potato, but the addition of potato to broth forms a more favourable medium than broth alone. Exposure to a temperature of 56·5° to 57° C. for ten minutes destroys the vibrios, but they are not killed in half that time.

Infection.—It appears probable that in the vast majority of cases natural infection occurs by the mouth, although the possibility of infection per vaginam must not be lost sight of. Experimentally the disease can be conveyed by the mouth and by parenteral injection. There is reason to believe that the vibrios can maintain their vitality and virulence for very considerable periods outside the animal body, but the length of time has not yet been definitely established. There may be an interval of from 13 to 113 days between infection and abortion.

Symptoms.—As in the case of bovine abortion there do not appear to be any definite symptoms until just before the abortion takes place. In animals in an advanced stage of pregnancy the udder is enlarged and the vulva swollen and discharging a sanious, mucoid material. In experimental animals this discharge has been noticed several weeks before abortion, and even a few days after infection.

Lesions.—The lesions are confined to the uterus, which, however, shows little or no alteration externally.

A considerable period often elapses between the death of the fœtus and its expulsion, and as a result secondary infection of the uterus with putrefactive organisms is common.

The posterior third of the organ is found to be somewhat thickened and œdematous, and on section exudes a yellowish fluid. In the early stages of the disease the mucous membrane may appear normal, but later it is congested and there is a quantity of hæmoglobin-stained mucoid exudate between the uterus and the chorion. The cotyledons are congested and soft, and some may be found separated.

Unless contaminated by putrefactive organism the exudate is odourless.

Diagnosis.—The determination of the presence of the vibrios by microscopic examination of smears from the expelled membranes, especially the cotyledons, and the contents of the fœtal stomach, confirms the diagnosis. The apparent absence of the specific organism, however, does not establish the absence of the disease. When the contents of the womb are not contaminated with other organisms cultural methods may be employed as an aid to diagnosis.

Agglutination Test.—The serum of infected animals agglutinates suspensions of the vibrios in dilutions up to 1 in 1000 and possibly higher, while that of non-infected animals fails to produce clumping in dilutions of 1 in 10 to 1 in 25.

Immunity.—Some individuals appear to be more resistant to infection than others, but no particular breed is naturally immune. Possibly one attack of the disease confers some immunity, since it is unusual for it to appear in two successive years in the same flock.

Treatment.—*Curative.*—There is some hope that treatment by means of an anti-bacterial serum may be of considerable value.

Preventive treatment should be carried out on similar lines to those which apply to abortion in cattle. All contaminated material should be destroyed or disinfected, and all infected membranes, discharges, and fœtuses should be burnt. Animals which have aborted or are suspicious should be isolated, and apparently healthy animals should be moved on to clean ground wherever practicable. The genital passages should be irrigated with weak antiseptic solutions, such as 3 per cent carbolic acid in water, 1-2000 corrosive sublimate, or a ½ per cent solution of lysol in water, and the soiled fleece should be disinfected.

J. B. B.

CONTAGIOUS ABORTION IN MARES

During the past thirty years numerous observations have been made regarding the etiology of contagious abortion in mares in Europe and the United States of America. Various types of organisms have been described as the responsible agent. McFadyean and Edwards have recently confirmed the findings of several previous observers (de Jong, Good, and Corbett and others) and have established the fact that contagious abortion of mares is a specific disease.

Symptoms.—As in the case of contagious abortion of cattle and sheep the premonitory symptoms are not well marked or diagnostic, and the observer can only be guided by the previous history of the stud. More abortions occur during the eighth month of pregnancy than at any other time, although occasionally mares have been observed to abort during the early stages (Good and Corbett). Mares which are infected but carry their foals for 10½ months usually deliver them alive, but many of these animals, although apparently healthy when born, subsequently die from pneumonia, while others develop a fatal white diarrhœa. A large number of mares in a stud may abort one year and the majority will produce healthy foals the following season. Abortions in the later stages of pregnancy are accompanied by but little effort or subsequent ill-effect, although death due to metritis sometimes results.

Bacteriology.—*B. abortivo-equinus* has a marked tendency to pleomorphism both in the animal body and in artificial cultures. The predominant form is that of a coccus or short plump bacillus of from ·5 to 1 micron in length and ·3 to ·5 of a micron in width. Distinct rod-shaped elements are usually also present. When cultivated in artificial media there may be some increase in size of the organism. It stains irregularly with the basic aniline dyes, sometimes showing bi-polar staining, and is Gram-negative. It grows upon the ordinary media under aerobic conditions. The growth upon agar is characteristic. After two to three days' incubation the surface growth shows a peculiar wrinkled appearance, which resembles tanned alligator or lizard skin. The peculiarity is only seen in rich cultures of the organism (McFadyean and Edwards). *B. abortivo-equinus* is motile and does not form spores.

Experimental Infection.—Good and Corbett found that the intravenous injection of a suspension of the organism into a rabbit produced death in one hour. Subcutaneous injection of the stomach contents of an aborted foal into pregnant guinea-pigs produces abortion in 36 hours. In pigs the intravenous injection of broth cultures produced a transitory malaise from which the animals recovered in from three to four days. Abortion was produced in a pregnant ewe in 72 hours and in a sow in 41 hours. The intravenous injection of the organism into pregnant mares produced abortion, and the organisms were recovered from the chocolate-coloured discharge up to five days after the abortion.

Lesions.—The disease is a specific metritis, and the abortion is a symptom. There is a chocolate-coloured discharge from the vagina with a characteristic "sour" odour. This odour is also evident in the fœtus and membranes, and is more marked in some cases than in others. The stomach contents of the fœtus at first give an acid and later an alkaline reaction. The lungs may show hepatized areas, the spleen is usually enlarged, and together with the liver may show hæmorrhages upon its surface. The mesenteric lymphatic glands are congested.

Diagnosis.—The history of the case and the nature of the discharge afford some help in the diagnosis of the disease. The causal organism may be demonstrated microscopically in many cases in smears from the exudate and the membranes. Cultures may be obtained in artificial media from otherwise uncontaminated material (discharges, membranes, heart-blood, and alimentary canal of the fœtus).

Agglutination Test.—Good and Corbett have shown that the serum of normal horses agglutinates suspensions of *B. abortivo-equinus* in dilutions of from 1-200 to 1-300, while that of infected animals agglutinates similar suspensions in dilutions of from 1-500 to 1-5000.

Complement Fixation Test.—Surface found that the complement binding titre of the blood of animals suffering from contagious abortion varies from ·1 to ·01, while that of normal horses gives a negative reaction in these quantities.

Treatment.—Probably but little can be done beyond the strict isolation of suspicious cases and the thorough disinfection, and when possible destruction, of contaminated materials.

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J. B. B.

WHITE SCOUR IN CALVES

The term white scour indicates the chief symptom of a serious disease of young calves. It is a form of diarrhœa occurring usually within a fortnight after birth and is due to organisms of the colon type which may be found in the blood and organs of affected calves. Infection usually takes place by way of the mouth, although it is probable that in some instances infection occurs through the unhealed navel immediately after birth. On the other hand, it has been asserted by Nocard and others that the cause of the condition is an ovoid organism of the Pasteurella type and is infected solely through the navel. The weight of experimental evidence, however, very strongly supports the former view.

The disease runs a rapid course and is fatal in a very large proportion of cases. In those cases, however, where recovery from white scour occurs certain secondary infections are likely to arise, such as pneumonia or arthritis, etc., which often prove fatal. Those surviving remain weakly for a considerable time.

Symptoms.—These may occur within a few hours after birth or may not develop for several days or even up to ten days or a fortnight. In calves that show the symptoms at a very early stage it is probable that infection took place either before or during birth, and in such cases death may occur on the same day or within the next day or two. A very liquid diarrhœa is set up, which is generally yellowish-white, often curdy or frothy and sometimes blood-stained. The fæces are particularly offensive in odour. There is constant straining, grunting, moaning, and rapid emaciation. The perineum, tail, and often the thighs are badly soiled and may become excoriated with fæces. The eyes appear to be sunk into the head, the sub-orbital fat being absorbed, and the patient presents a very miserable appearance with belly tucked up, arched back, the hair harsh and lacking in lustre, and hide-bound condition of the skin. The temperature in the early stages is high (105°-6° F.), but as the condition advances it becomes lower and even subnormal, while the extremities become very cold and the patient collapses and is unable to stand. Occasionally brain symptoms may be seen, such as blindness.

Before death in the protracted cases complications may set in, the joints may swell, while pneumonia and necrosis of the mouth may develop.

Treatment.—Curative measures are not of much service. In the stronger calves internal antiseptics such as quinine, salol, tannoform, and hyposulphite of soda have all been given and good results claimed for them, while astringents such as tannoform and chlorodyne given several times a day may assist in checking the diarrhœa. Careful attention should be paid to the feeding. When the calf is suckling the udders and teats of the cow should be frequently wiped and disinfected in order to avoid swallowing any infected material, while if the calf is being hand-reared suckling utensils should be scalded or steamed.

Affected calves should be placed in clean, light, well-ventilated and dry surroundings and not, as is so often the case, placed into dirty, dark, badly-drained calving pens, sometimes up to their knees in manure.

Preventive Treatment. — Prevention is far more likely to yield satisfactory results. It consists primarily of the abolition of calving boxes and thorough disinfection of the place in which the calf is to be born and cleanliness at the time of birth, special attention being paid to the frequent changing of bedding. The navel should be ligatured and disinfected at birth and periodically disinfected, especially on premises where the disease is known to exist. The external genitals of the cow should be disinfected before parturition. A protective serum has also been produced by the use of organisms of the coli and paracoli types. As it is possible that several different types of organisms may be responsible for the affection polyvalent serum should be adopted. Good results have followed the use of this serum when injected subcutaneously immediately after birth on farms where the disease is known to exist.

CONTAGIOUS PNEUMONIA OF YOUNG CALVES

This is an infectious form of pneumonia of calves which may come on primarily or occur as a sequel to white scour. It frequently makes its appearance in from two to three weeks after birth, and calves up to two or three months old may become infected.

Anatomical Changes.—Both lungs are usually found affected with a diffuse pneumonia, and in the affected areas are small necrotic patches with a tendency to abscess formation. In some cases there is a more or less marbled appearance of the lung which has caused confusion with contagious bovine pleuro-pneumonia, but the age of the patient and the acute nature of the affection should readily serve to distinguish it. There is often pleurisy with an exudate in the chest and coagulated lymph on the pleura. In less acute cases there may be adhesions of the lungs to the chest wall.

The necrotic lesions of this affection usually contain large numbers of the Preisz-Nocard bacilli, the presence of which, together with the absence of lesions in the lymphatic glands, serves to distinguish the affection from tuberculosis.

Symptoms.—The calf is very dull and depressed and rapidly loses condition. It refuses to suckle, while the bowels are irregular but most frequently diarrhœic. There is considerable coughing and the respiration is blowing, the mucous membranes are brightly injected, while the temperature may be up to 105° or 106° F. On auscultation consolidated areas are detected in the lungs and in many cases the friction of pleurisy. A number of calves are usually affected at the same time, and the disease is fatal to a large proportion of them.

Treatment.—First adopt thorough disinfection principles ; take away the healthy calves, and to the affected administer stimulants and internal antiseptics such as quinine, salicylates, and potassium iodide. The hypodermic injection of nuclein may also be helpful. Counter-irritants or stimulating liniments should be applied to the chest wall and medicated inhalations as with carbolic acid or eucalyptus are advisable. All suckling pails should be scalded and thoroughly disinfected, while the best hygienic conditions possible should be provided, such as a clean, warm, well-ventilated house and a plentiful supply of clean dry bedding.

JOINT-ILL

Joint - felon ; Polyarthritis, and Rheumatic-arthritis of young stock ; Navel-ill ; Omphalo-thrombo-phlebitis.

This is a disease occurring in young stock, due to infection by way of the umbilicus, and occasionally before birth. It is characterized by abscess formation at the umbilicus, lesions in various joints, most frequently the stifle, hock, and knee, and frequently abscesses in some of the internal organs.

The condition occurs principally in foals, calves, and lambs, and may be produced by a variety of organisms. In the lesions have been found streptococci, staphylococci, ovoid organisms of the so-called Pasteurella type, colon bacilli, necrosis bacilli, and also in foals the same organism that causes contagious abortion of the mare (*B. abortivo-equinus*). The position and character of the umbilicus render it particularly easily soiled, and when bacteria have found a lodgement, the warmth, moisture, and pabulum, both of the tissues and of the clot forming in the vessels, afford almost ideal conditions for their growth, and this results usually in the development of an abscess or abscesses. Access to the umbilical veins is then easy, and pyæmia may follow, with lesions in the liver, lungs, kidneys, and the joints, which latter appear in many cases to be predilection seats of the organisms. Organisms may also extend along the urachus to the bladder and set up cystitis. The invasion of the urachus tends to retard its normal closure, with a resulting prolonged moist umbilicus favouring still further infection. Thus a primary umbilical infection may cause pervious urachus, while a primary pervious urachus obviously predisposes to navel infection.

It has been suggested that infection may be hereditary in that the succeeding progeny of some mares become affected, and this view is somewhat supported by those few reported cases in which the joints of foals are enlarged at birth, the result of intra-uterine infection. Although that may occur, a more common hereditary feature is that all the progeny of certain dams have a very slowly cicatrizing umbilicus due to a rather large umbilical ring, and obviously such an hereditary condition must be regarded as a predisposition. Another reason for progeny developing the condition year after year is frequently that they are born in the same old places without proper care having been taken to disinfect them. In fact that is the case to such an extent that one might almost regard the disease as one of old foaling and calving boxes and of old lambing folds. I have known a farm where year after year most of the foals developed joint-ill. They were all born in the same big, roomy loose box, the only attempt at disinfection being occasional white-washing of the walls. Good results were only obtained when the farmer, who would not pay attention to detail, was persuaded to allow his mares to foal out at grass as the lesser of two evils. This was immediately successful, and the mares which were thought to be transmitting it were proved " not guilty."

Symptoms.—Symptoms vary according to the degree of virulence of the organism. In a few cases joints have been found swollen at birth. In the ordinary case symptoms may occur in about a week or ten days after birth up to as long as six months or even longer.

The first signs are usually those of general depression and indisposition to suckle or feed. The temperature is elevated 2° to 4° F., breathing rather hurried, stiffness of movement, joints somewhat swollen ; tendency to lie down rather excessively. The bowels are irregular, sometimes diarrhœic, sometimes constipated. Loss of condition is very rapid. On examining the umbilicus it may be found to be wet and discharging either a little moisture or even urine, or it may be dry externally and swollen, owing to abscess formation. In other cases where the joint lesions are late in developing there may be no umbilical lesions discoverable.

On examining the joints, some of them are found to be swollen, hot, tense, and painful, with a firm gelatinous consistency of the surrounding connective tissue and fibrous structures, due to œdema. These may then develop fluctuations in different parts of the joints, and bursting, reveal the existence of abscesses, and liberate a mixture of blood-stained pus and synovia with shreds of tissue. The diffuse condition is the predominant form in foals, and the stifle the commonest seat; abscess formation appears to be commonest in lambs, while in calves the two conditions are met with about equally.

Prognosis is not very favourable, as the condition is fatal in about 50 per cent of cases ; death may occur in two or three days or two or three weeks. The severity appears to be in direct ratio to the rapidity of the onset after birth. Complete recovery may occur in a number of cases, but in most recoveries the patients remain unthrifty for a long time. In other cases the joints remain permanently enlarged and may cause chronic lameness—a condition obviously more serious in foals than in other species.

Treatment.—As will readily be assumed from the preceding paragraph, curative treatment is far from satisfactory.

Externally. The first thing to do is to examine the umbilicus, and if any abscess is found it must be well opened and evacuated, and thoroughly disinfected with such potent agents as a single dressing of carbolic acid and glycerine in equal parts, and subsequently with less severe dressings of either phenol, iodine, or corrosive sublimate. The treatment of the joints must vary according to the condition. If abscesses are present they must be evacuated and irrigated with disinfectants, and then injected with clove oil and bandaged. In the non-suppurating joints, a liniment may be applied, consisting of lin. camph. ammon. 1 part, lin. bellad. 2 parts, lin. saponis 3 parts ; or a weak blister of biniodide of mercury ointment (1 : 12 or 16) applied.

Internally. Stimulants and internal antiseptics are indicated. The latter may consist of either potass. iodid., hydrarg. biniod., salol and other salicylates, quinine, sodæ hyposulph., and tannoform. Hypodermic injections of nuclein are also of service, while collargol and ichthargan have also been recommended intravenously.

Various vaccines and sera have been put upon the market for the treatment of joint-ill, but in the case of calves and lambs their use is likely to be very disappointing, since the affection may be brought about by any of a large variety of organisms acting either singly or in combination. No stock vaccine prepared from any individual organism is very likely to give satisfactory results in the present stage of knowledge, though a polyvalent vaccine may prove serviceable. In the case of the foal a vaccine prepared from the organism of contagious abortion of the mare was found by McFadyean to be useful in a number of cases.

Prevention.—Although curative treatment is so unsatisfactory, fortunately preventive measures are comparatively easy and highly satisfactory. As already noted, the affection is largely one of houses and folds. Consequently the old style of foaling and calving boxes must be abolished, and if special boxes are to be used they must be constructed on hygienic principles, so that they can be properly lighted and thoroughly ventilated, drained, and disinfected. Thorough and periodically repeated disinfection, paying especial attention to the floor, is essential. Lambing folds must be destroyed each year after the lambing season is finished, and reconstructed on different sites in succeeding years. In each species the progeny must be received on fresh and unsoiled straw, and the umbilicus thoroughly dressed with a potent disinfectant. The cord must be ligatured with antisepticized material close to the abdominal wall (*i.e.* about half an inch away) and then cut off about an inch below. A disinfectant should be applied daily in the case of foals until the umbilicus is cicatrized, and in the case of calves and lambs so far as is practicable. A wide range of disinfectants is available, and so long as they are *really* disinfectants there is little to choose between them. Amongst the best are 3 per cent to 5 per cent carbolic acid in aqueous solution ; 2 per cent lysol ; 1 : 1000 perchloride of mercury ; 1 per cent or 2 per cent solution of iodine in potass. iodid. sol.; zinc chloride 1 per cent, or zinc sulphate or copper sulphate 2 per cent sol.; salicylic acid 2 per cent. If I have any choice of these it is for the metallic salts solutions, which have a tendency to harden the stump and so reduce the liability to infection.

In studs and farms where the disease is known to exist rather more detailed measures should be adopted ; as, for example, the disinfection of the external genitals of the dam, the receiving of the newly-born on a clean sheet, and the sealing of the navel in the manner suggested by Nocard. This consists of the application *seriatim* of three dressings. The first is made up of iodine ʒss., pot. iodid. ʒi., and rain water 1 litre (about 1¾ pints). This is swabbed round the parts with a large soft brush, and is followed by the second solution, which consists of iodine ʒss. in methylated spirits 1 litre, applied by another brush. This spirituous solution is intended to dry the navel, and after it has evaporated the third dressing is applied : this consists of iodine 1 per cent dissolved in collodion. This process sounds rather complex, but in reality it is quite simple if done methodically. The solutions can all be placed in glass jam jars and labelled No. 1, No. 2, and No. 3 respectively, and the handle of the brush in each case passed through a hole in the cork. The jars are kept at hand at the time of the birth, and as the newly-born is held on its side or back for ligaturing the umbilical cord the dressings are very easily applied. There is no doubt that some of the less intelligent farmers and herdsmen will not be persuaded to undertake the process, and the nearest simple approach to it consists in the application of Stockholm tar to the umbilicus after ligation and disinfection.

One must not forget the calf sheds so largely employed on dairy farms where the calves are often separated at birth from the mothers. These places are frequently of the filthiest description, dark, undrained and unventilated ; the calves often standing up to their knees and hocks in wet material, half litter and half excreta.

Then again, on infected premises attention must be paid to the possibilities of infection by the mouth. Mammary glands of the dams should be kept clean, and all feeding utensils such as suckling pails, etc., constantly steamed or scalded. If any animals become affected they must be immediately removed from contact with the healthy : no abscesses should be opened in the place where other animals are kept, and evacuated pus should be destroyed by burning. A separate attendant must be told off to look after the affected animals.

Peggie (of Biggar, Lanarkshire) has reported (1920) excellent results from the use of a polyvalent antistreptococcic serum. Assuming that some of the cases of joint-ill in foals were due to ante-natal infection and others possibly post-natal, he decided to conduct prophylactic treatment along two main lines, viz. (1) to treat the mare during pregnancy, and (2) to treat the offspring immediately after birth. In one season he treated sixty in-foal mares, injecting 14 c.c. of the serum ten days before the mares were due to foal and repeating the dose a week later. The foals received 7 c.c. 24 to 48 hours after birth. Not one case of joint-ill was observed in the foals of the injected mares, though seventeen of the mares had during previous seasons given birth to foals that had developed joint-ill. The mares received no other special treatment, but antiseptic tape was used for ligature of the umbilicus when necessary, and the cords were dressed with tincture of iodine. A number of mares in the surrounding district were not injected, and the offspring of four of them developed fatal joint-ill.

The condition is a serious one, for it entails enormous losses, and it is only by tackling it seriously that it can be controlled. It *can* be controlled, but only by adopting stringent preventive measures, and not relying upon curative treatment.

G. H. W.

INFECTIVE MUCO-ENTERITIS OF CALVES AND FOALS

This is a form of diarrhœa which attacks calves between the ages of a fortnight and three months, but may be met with in older animals. It is commonly called " Scour " or " Skit," and is regarded as a different affection from white scour. It is highly probable, however, that it is caused by organisms of the same type and is even possibly the same disease. The main difference is in the symptoms and mortality, being due to the fact that the calves are a little older at the time of infection and are therefore probably more resistant.

Infection takes place by the mouth, and as in white scour soiled teats, dirty suckling pails, and contaminated foods are the media of infection.

A rather important point supporting the view that this affection is different from white scour is the fact that the disease may exist on a farm for years and during that period not a single case occurs in the very young calves of from two to ten days old as is commonly the case in true white scour. Certainly if the two affections are identical both forms would be expected more or less simultaneously on a farm.

A form of scour in calves of this age may also be due to dietetic errors in artificially fed calves.

Symptoms.—The most prominent symptom is diarrhœa, the fæces being either yellow or clay-coloured and extremely offensive in odour. It causes soiling of the tail and perineum and may produce excoriation of the skin. The appetite is capricious, the calf feeding well at some meals but refusing others on the same day. There is great loss of condition, a pendulous abdomen in some cases but tucked up in others. The coat is harsh and dry and the condition of hide-bound is often set up. The mucous membranes are very pallid ; the temperature is usually elevated 3° or 4° F. in the early stages, becoming low or subnormal in fatal cases. It is fatal in a large proportion of calves and particularly so in newly purchased animals brought in from a non-infected herd. Animals that recover usually remain backward and unthrifty for a considerable time, even up to a year or more.

Treatment.—Treatment of this affection is not satisfactory. Preventive measures should be adopted on farms where it is known to exist. Warm, dry, well-ventilated, and drained boxes should be provided. Calving boxes should be very thoroughly disinfected and one would like to think they could be abolished as there is no doubt they are the hot-beds of infection.

If the calf is suckling the udder of the cow should be wiped before the calf is allowed to suckle her. That of course is unnecessary and impossible where the cow and calf are out at pasture. When fed artificially utensils should be cleansed with hot soda and water and then steamed, and all foods should be boiled before being given. Vaccines have been prepared for this condition and good results have attended their use in many cases.

Medicinally it is a good plan to commence treatment with castor oil ℥ii., and to follow with intestinal disinfectants and astringents. Amongst the best are tannoform, carbonate of magnesia, salol and hyposulphite of soda, while chlorodyne ʒi. will often check the diarrhœa. An old combination which has frequently been found useful, especially when the scour has been due to dietetic errors, consists of powdered opium gr. v., catechu ʒi.-ii. and prepared chalk ℥ss.-℥i. This may be administered twice daily in flour gruel.

CALF DIPHTHERIA (NECROTIC OR ULCERATIVE STOMATITIS OF CALVES)

Calf diphtheria is an acute contagious disease of young calves caused by the bacillus of necrosis and characterized by diphtheritic and ulcerative lesions of the mouth and throat. It has no association whatever with human diphtheria. Calves may become affected within a few days after birth, the vehicles of infection being contaminated food materials and suckling pails. The organism apparently remains alive for a considerable time in the sheds on dirty floors, etc., and is therefore easily transmitted to healthy calves. Infection is probably facilitated by injuries of the mucous membrane of the mouth and also by the eruption of teeth.

The period of incubation is about five days.

Symptoms.—Symptoms may be observed within a week of birth and commence with a capricious appetite, loss of condition, and weakness; the temperature is elevated 2° or 3° F. A day or two later there is a dribbling of saliva, coughing, a dirty yellow nasal discharge, and an inability to pick up any solid food. Any solids taken into the mouth are usually dropped out again. The bowels are irregular with a tendency towards diarrhœa. On examining the mouth yellowish patches may be seen on the tongue, the sides of the cheeks, and the roof of the mouth, and sometimes in the pharynx. In some cases quite a large area of the tongue may be affected, often beginning at the side a few inches from the tip, and sometimes almost half the tongue may be dead.

In acute cases death may occur in from four to five days. In the less acute cases the course may extend over several weeks and may then either terminate in death from pneumonia or in recovery after a long convalescence owing to the length of time necessary for the healing of the lesions in the mouth and throat.

On post-mortem examination the ulcers and necrotic lesions may be observed in the mouth and pharynx. The necrotic patches are yellowish in colour and somewhat raised above the surrounding tissues and are firmly adherent. The ulcers result from the detachment of the necrotic tissues and exhibit a red, angry-looking base with thickened borders. Similar lesions are occasionally seen in the nasal cavity and affecting the epiglottis. The lungs may also be involved, showing necrotic areas and sometimes pleurisy. At other times the intestines may be ulcerated. The liver of calves may also be attacked by the necrosis bacillus, but this is probably the result of a navel infection.

Treatment.—At once remove healthy calves from the infected houses, providing the best hygienic conditions possible in the way of ventilation, drainage, and thorough disinfection. The healthy calves must be examined daily and their temperature taken, and any showing elevation of temperature quickly removed from the rest. The affected calves must be isolated or segregated. The yellow necrotic patches must be removed either by a curette or a blunt knife and scraped so that all the deposit is removed. The part should then be painted daily with a strong disinfectant such as tincture of iodine or 5 per cent solution permanganate of potash; salicylic acid rubbed into the part is also very useful.

Internal antiseptics should be administered once or twice daily, such as potassium iodide gr. xxx., quinine gr. xx., acetyl-salicylic acid gr. xxx., or potassium chlorate gr. xxx. Stimulants are sometimes necessary, in which case carbonate of ammonia gr. xxx. and powdered nux vomica gr. xv. may be employed with advantage.

The patient's strength must be maintained by a generous supply of highly nutritious foods, such as milk and eggs, linseed or oatmeal gruel, which must be forcibly administered if the patient refuses to take them voluntarily. The suckling pails and all utensils must be scoured with soda and water and sterilized by steaming or with boiling water.

G. H. W.

BLACKLEG

Blackquarter; Symptomatic Anthrax; Quarter Ill; Quarter Evil; "Felon"; "Strike"; "Struck"; "Hyans"; "Irons"

Blackleg is an acute bacterial affection of the ox and sheep, caused by the bacillus of blackleg. Cases have been recorded in the horse and pig, but there is some doubt as to their authenticity. In cattle the common period of susceptibility is from six months to two years old, but it may occur in younger calves and in adult cattle. Young calves are not immune, and their escape is generally due to the fact that they are rarely exposed to infection in early life. The writer has had experience of malignant outbreaks in calves from one to two months old. Sheep of all ages are susceptible, and the writer has met with it in lambs ten days old. Rabbits are not susceptible to the affection, and therefore cannot be regarded as a means of spread from one field to another as may occur in anthrax. Blackleg is an enzoötic disease having a rather curious distribution; even on a particular farm one or two fields may be known to be infective, while the rest of the farm escapes. It has a very wide distribution throughout the world, occurring in all continents and all climates.

Etiology.—Blackleg is due to the bacillus of blackquarter (bacillus of Chauveau), an anaerobic organism commonly found in the soil as a saprophyte in infected areas. The organism is a rod-shaped bacillus longer than the anthrax bacillus but more slender. In the tissues of the living animal it forms spores which distend the bacillus and may either be in the middle giving it a lemon-shape appearance, or terminal producing a tennis-racket shape. It is mainly a tissue parasite, and is found in greatest abundance in the emphysematous swelling which is characteristic of the disease. It is there in the inflammatory exudate, in the substance of the muscular fibres, and in the connective tissue. This inflammatory swelling is the only constant seat of the organism. It occasionally invades the blood stream, but it does not occur in such numbers as to make it possible to find it by microscopic examination. When there are exudations in the serous cavities of the body it is usually found there.

Method of Infection. — There is still some doubt as to what is the natural method of infection. It is most probably caused by inoculation at the seat of the lesion. It is improbable, as has been suggested, that infection takes place through some wound of the foot, as lesions have never been found below the knee or hock. It is also stated that infection may take place as the result of feeding on infected food or drinking-water, but attempts to produce the disease in this way have rarely been successful, even when large quantities of virus have been administered.

The *period of incubation* is from two to five days, and the disease may run its course in from twelve to forty-eight hours ; in rare cases it may last a week, and in adult cattle even longer.

Symptoms.—In natural cases in calves the first thing noticed is a lameness which is frequently limited to one hind limb. The animal separates itself from the rest of the herd and refuses to feed, rumination ceases, and the animal lies down. There is blowing respiration, accompanied by grunting. The temperature is elevated and may be anything from 104° to 107° F. On closer examination a local swelling may be discovered, most often in the upper part of the hind limb, at other times in the shoulder, the neck, or in the abdominal wall. This is at first small, hot, and painful, but rapidly extends, involving a large area. Later the swelling becomes cold and painless. On manipulation it is found to be emphysematous and crackles something like thick parchment. If cut the part is found to be engorged with dark-red bloody exudate which may be more or less frothy. The lymphatic glands associated with the part are swollen and hot. As the condition advances the breathing becomes more laboured and the patient becomes prostrate, the temperature falls to normal or slightly subnormal, to be quickly followed by death. In the rare non-fatal cases in cattle a deep-seated abscess forms which is very slow in healing, causing great loss of condition and emaciation. An animal so affected is not an economical asset, and is best destroyed.

In sheep the disease is not so commonly met with, although it is probably far more common than is usually suspected. It runs a much more rapid course than in cattle, and not infrequently affected sheep are found dead without symptoms having been observed. In this respect it more closely resembles anthrax. If seen before death the sheep are found to be very stiff and become rapidly weak and unable to stand. They then become cast on their backs and kick, and soon die. The local lesions are rarely so large as in cattle, and are not so markedly emphysematous. They are most often met with affecting the neck, the breast, and sometimes in young lambs involving the abdominal floor in the region of the umbilicus.

Anatomical Changes. — The characteristic lesion of blackquarter is an emphysematous swelling crackling on manipulation. In sheep the wool over the affected part is easily pulled out. When the swelling is cut into, the muscles and other tissues are dark red, or almost black, and have a more or less porous appearance, and are rather dry-looking. This is due to separation of the fibres by gas produced by the organisms. In other parts there is a blood-stained exudate and hæmorrhage into the muscular tissue. The affected tissues have a characteristic sour odour, somewhat resembling rancid butter, and in early cases not offensive or putrid. The lymphatic glands of the part are swollen and often hæmorrhagic. Sometimes there are blood-stained exudates in the pleural and peritoneal cavities. The carcase rapidly becomes tympanitic after death as in anthrax.

Treatment.—Curative treatment is useless. The old treatment consisted in incising the lesion deeply and rubbing in strong disinfectants, such as tincture of iodine, carbolic acid, and perchloride of mercury, while internally carbolic acid, quinine, and other internal antiseptics were administered. Such treatment is to be condemned on account of the spreading of the organisms, which are very abundant in the liquid escaping from the cut surfaces. Moreover, from the economic standpoint results do not justify treatment.

Preventive Treatment.—As the disease is frequently limited to certain fields and districts, cattle of susceptible age should be kept off them. Such pastures may be dressed with lime, or if possible should be ploughed up. Good results are claimed from the periodical dosing of calves and yearlings with alteratives such as hyposulphite of soda and flowers of sulphur, thus maintaining the high standard of health and allowing them to resist infection. An old method of prevention consisted of inserting setons or rowels in the dewlap and dressing them with an irritant such as turpentine liniment, hellebore, or mild blistering ointment. Although good results were claimed for this procedure, it is more than doubtful if it had any beneficial effect.

Attention should be paid to the proper disposal of a dead carcase, either by cremation or burying deeply with quicklime. If the carcase has been cut, all discharges which may have escaped must be similarly treated. The area in the field round about where the carcase lay should be thickly covered with quicklime. The adoption of these principles will check the disease, but the only method of ensuring protection is the resort to vaccination.

Protective Inoculation against Blackquarter.—Several methods of vaccination against blackleg have been practised, each of them affording good results.

(1) *Arloing's Method.*—This consists of the inoculation of two vaccines prepared from muscular tissue from a perfectly fresh blackquarter lesion by exposure to heat (dry heat). The first vaccine is the more attenuated and is designed to prepare the animal for the second rather stronger vaccine. The first vaccine is inserted under the skin of the tail 5 or 6 inches from the tip, and 8 or 10 days later the second vaccine is inserted several inches higher. The vaccine is sometimes inserted at the shoulder. Full antiseptic precautions must be taken. Immunity conferred by this method is sufficient to carry the animal through the period of natural susceptibility. The best age for vaccination is between 6 and 8 months.

(2) *Kitt's Method.*—This method consists of the inoculation of a single vaccine also prepared from dry muscle but by moist heat and administered in larger doses than by the former method. This method entails less labour than Arloing's but is probably not so effective, while at other times it is attended with greater danger.

(3) *Leclainche and Vallée's Method.*—This consists of two injections; the first is a hyperimmune serum, the second consists of attenuated pure culture. There is an interval of a week between the two injections, which may be made in the shoulder or the tail. Nitta asserts in opposition to Leclainche and Vallée that a mixture of immune serum and virus in proper proportions may be successfully employed, thus reducing the two operations to one.

(4) *Nitta's Method.*—This method consists of the injection of 5 to 10 c.c. of a filtrate of a pure culture of the blackleg organism. The seat of injection is the chest wall and only one operation is necessary. Nitta claims as additional advantages of the method that it is inexpensive and that it is free from all danger as the filtrate is quite germ-free.

In the case of both Arloing's and Kitt's methods the vaccine is put up in various ways by different laboratories. Probably the best method consists of putting up the powdered vaccine in a dry state in a sealed glass tube, to be mixed with sterile water for injection. The other methods consist of enclosing the vaccine in a pellet, or impregnating a little wool or fine tape with the vaccine, and these are introduced under the skin by special instruments supplied with them. The danger following protective inoculation against blackleg by either of these methods is infinitesimal although occasionally animals succumb, possibly through being in the incubative stage of the disease at the time of inoculation. Sometimes it is due to a hypersusceptibility on the part of the individual, while at other times it may be due to the vaccines having unaccountably resumed virulent characters.

G. H. W.

ANTHRAX

Splenic Apoplexy ; Splenic Fever ; Milt Fever

Anthrax is the disease caused by a specific organism, the *Bacillus anthracis* or the anthrax bacillus. In warm and fairly hot climates the bacillus is capable of living and multiplying outside the animal body, but in the British Isles this is highly improbable, the temperature rarely being favourable. Spores, however, will remain alive in the soil in temperate and cool climates for long periods.

The disease usually assumes an enzoötic form, multiple cases being due rather to a common source of infection than to actual spread from animal to animal.

Susceptibility.—All domestic animals are susceptible, the ox being most commonly affected, and then the sheep, horse, pig, and dog in order named. Outbreaks are sometimes serious in elephants in Burma. Birds as a class are highly resistant to natural infection, but the ostrich is not uncommonly attacked. Man is also susceptible, becoming infected secondarily by handling affected carcases or hides and offal from such (malignant pustule), and also by the inhalation by woolsorters of spores from imported wool.

Sources of Infection.—As already mentioned, in hot climates the bacilli may be living in the soil, or in cooler climates spores may be present. In South Africa Robertson and Andrews have separately found the actual organism in the water supply. If conditions are favourable to the life of the organism in the soil it has been noticed that in seasons when " keep " is scanty more animals become infected—presumably through feeding closer to the ground with consequent ingestion of a considerable amount of soil.

The disease is directly inoculable through wounds and abrasions, and it is in most cases by wound infection that man is affected. In the great majority of veterinary cases the disease is produced by ingestion of infected material. Fodder or feeding cakes may introduce anthrax either by virtue of having grown on contaminated soil, or, as is more general, through having been transported in vehicles or holds of ships which have previously carried hides or offal from anthrax carcases.

Opening or skinning an infected carcase will set the organisms free and allow them to sporulate, increasing the risk of subsequent infection of other animals and man. Again, infected carcases left to be devoured by pigs or wild animals are a source of danger for the same reason.

Symptoms.—Owing to its rapid fatality, symptoms are often unobserved, the animal being found dead. This sudden death is in itself suspicious of anthrax. If observed, the symptoms depend on the seat of infection, usually either the throat or the gastro-intestinal tract. Different animals present different symptoms.

In the ox a very acute, rapidly fatal enteritis appears, so rapid that death may supervene before any symptoms have been observed. In other cases there is great and continuous pain, high fever, with the temperature up to 107° or 108°, and sometimes delirium. Later, sudden collapse is followed by rapid death.

In the sheep the course is more rapid. Few, if any, symptoms are seen, especially as the sheep is an animal on whom individual observation is rarely expended.

In the horse the course is rarely so rapid as in the ox, and symptoms are usually observed; violent and continuous colic, high temperature, straining and subsequent collapse and death. A throat form is also common, in which case there is a painful, rapidly growing swelling of the throat. Breathing becomes difficult and the horse becomes frantic with distress. Death supervenes, usually directly attributable to asphyxia, although at or about the point of death the bacilli may become generalized, but not invariably.

In the pig and dog the throat form is by far the commoner although in the pig anthrax may take a subacute, general form with fever lasting several days.

In the ostrich symptoms are indefinite. Apparently healthy birds die after a few hours' dulness followed by prostration. In other cases they may remain alive for a day or two with indefinite symptoms. In any case high temperature is not so invariable as in the domestic mammals.

Post-mortem Appearances.—In the case of animals suspected to have died of anthrax no general post-mortem examination should be made owing to the danger of contaminating the soil by spilling blood in which the bacilli are present in large numbers. An examination may be performed inadvertently, however, in which case the lesions cannot fail to suggest anthrax.

The dead carcase rapidly undergoes putrefaction with formation of gas and distension of the abdomen. The rectum and vagina are often everted, and blood may be forced through the external orifices. In throat cases the throat region is markedly enlarged and firm.

The first incision shows the superficial veins to be engorged with thick, black blood which does not clot properly, even if exposed for a long time. Inside the abdomen the lesions are of acute hæmorrhagic gastro-enteritis, the bowel wall being thickened and infiltrated with a yellow gelatinous fluid. The serous membranes show numerous small hæmorrhages, the lymphatic glands are enlarged, infiltrated and hæmorrhagic and black bloody material is often present in considerable quantity in the lumen of the bowel. The spleen, in the ox especially, is enormously enlarged, up to ten or fifteen times the normal size, and the spleen pulp is almost fluid. If cut into, the pulp is seen to be black and tar-like. This great spleen enlargement is not so pronounced in the pig, and considerably less pronounced in the horse.

In throat cases, as seen in horses and in swine, the lesions may be entirely confined to the throat region. There is great swelling and enlargement of associated glands, usually hæmorrhagic, and the whole region is infiltrated with a gelatinous yellow fluid similar to that which infiltrates the bowel. Small hæmorrhages may be seen, but are often overlooked.

The ostrich, although not presenting the same clinical symptoms as mammals, shows, generally speaking, the same lesions on post-mortem examination.

Diagnosis.—For absolute diagnosis the *Bacillus anthracis* must be recognized. Smears from any organ will usually do for examination, but as a post-mortem examination is rarely made, a blood smear only is examined for the presence of the bacilli. In the horse and sometimes in the pig one may have to examine the pharyngeal lymphatic glands or even the mesenteric glands before a diagnosis can be given definitely. If a blood smear is taken, any superficial vein is punctured, usually an ear vein.

Bacteriology.—The *Bacillus anthracis* is a comparatively large, rod-shaped bacillus, 4 or 5 microns long and 1 to 1½ microns broad. Commonly chains of them are seen, the elements being placed end to end, with square-cut extremities and very evenly segmented. The most distinctive character is the possession of a well-developed capsule.

Although the bacillus stains well with any of the common dyes and by Gram's method, probably the best method is that of McFadyean. The smear is lightly fixed by heat and stained with ordinary methylene blue. The bacilli then appear stained blue with square-cut or even slightly concave ends and the capsule stains pink. Even a naked-eye inspection of the stained smear shows a distinctly pinkish tinge, not observed in similarly stained blood in any other condition. If spores have formed they appear as unstained spots in the centre of the bacilli.

In cases of doubt, laboratory animals—preferably guinea-pigs — may be inoculated. If anthrax is present the animal dies and the bacillus is easily demonstrable in its blood.

Cultivation shows the bacillus to be aerobic. Absence of air will prevent its growth and also prevent the formation of the highly resistant spores which render the extinction of anthrax so difficult.

Control of the Disease.—Treatment is useless. One's energies must be confined either to preventing the occurrence or limiting the spread of the disease. In the British Isles control is confined to preventing further spread, whereas in South Africa and some other countries, particularly tropical and sub-tropical, the menace is so great that preventive vaccination is adopted.

In the former case the disease, being a scheduled disease, must be notified to the local police. The Local Authority inform the Medical Officer of Health and cause a veterinary examination to be made. In confirmed cases all movement of animals is prohibited in a prescribed area until the Local Authority is satisfied that no animal remaining is diseased. Moreover, milk of any diseased cow on the premises may not be removed. Carcases must be buried *in their skins* at a depth not less than six feet below the surface of the ground or must be burnt ; in either case under the direct supervision of the Local Authority. Litter, dung, fodder, etc., which have been open to contamination must be similarly dealt with. If a carcase has to be removed for burial, etc., all natural orifices must be plugged with tow or other suitable material soaked in strong carbolic solution, but in no case must the carcase be cut into except as much as may be necessary for a microscopical examination.

All buildings, pens, utensils, etc., must be disinfected thoroughly, also any vehicle used in carrying a carcase ; the disinfectants recommended being :

(*a*) Limewash of quicklime and water, containing at least four ounces of chloride of lime to the gallon.

(*b*) Limewash containing half a pint of carbolic acid to the gallon.

Preventive Inoculation.—In countries where protective inoculation is practised, as in South Africa, similar orders are enforced for the disposal of carcases, litter, etc., and in addition exposed animals are vaccinated. The common method of vaccination (Pasteur's original method) consists in injecting subcutaneously two doses of attenuated living anthrax bacilli, a first small dose being followed after 12 to 18 days by a second larger one. Sometimes a single dose only, of intermediate strength, is administered. The vaccination may cause occasional deaths and is consequently not recommended unless there has been anthrax in the vicinity. In that case the evidence to hand tends to show that this vaccination is of undoubted value in lowering the percentage mortality, and is therefore economical.

S. C. J. B.

TUBERCULOSIS

Consumption ; Phthisis ; Grapes ; Tabes Mesenterica ; Scrofula

Tuberculosis is a contagious disease affecting various species of animals, caused by the *Bacillus tuberculosis* (the bacillus of Koch). The disease is characterized by nodular lesions which may occur in almost any part of the body and having a tendency to undergo necrosis and caseation, sometimes followed by calcification.

Amongst the domesticated animals tuberculosis is far more frequently met with in the ox than in any other species, and of adult dairy cattle in Europe it has been estimated that at least 30 per cent are tubercular. The proportion rises steadily with the age of the animal, and probably not more than about 1 or 2 per cent of bovines under one year old are affected. It is commonest in *old dairy cows*.

The disease may be regarded as one of domestication, the housing of animals together greatly facilitating the spread of the disease. That is the reason why it is so much more common in dairy cows than other types, while it is very rare indeed in animals kept in the open, such as feeding-cattle, and it is practically unknown amongst wild animals in a state of nature. That these animals, including monkeys, are not immune is shown by the fact that when kept in confinement along with other animals, and thus exposed to infection, they readily contract the disease.

After cattle, swine are the most frequently affected, and next follow the horse, dog, and cat, and poultry. Sheep and goats are rarely affected. This is not due to any immunity on their part, but to their mode of life and the non-exposure to infection.

Etiology. — Tuberculosis is caused by the tubercle bacillus, which is a small bacillus, and is only capable of growth in natural circumstances within the animal body, although it may exist outside the body for a varying period according to the conditions of exposure. This emphasises the fact that tuberculosis is purely a contagious or infectious disease, and is due solely to the transference, either directly or indirectly, of the bacillus from an affected animal to a healthy one.

The organism is a slender non-motile bacillus sometimes slightly curved, and in the tissues is often found either singly or in groups like a bundle of faggots. At other times, in special cells (giant-cells) of the lesions, they may be grouped in a spherical mass or be arranged radiately in the cell. The organism is acid-fast, and is best demonstrated after staining by the Ziehl-Nielsen method, which consists of exposure of film preparations for ten or fifteen minutes to warm carbol-fuchsine 1 per cent, and then for a few seconds to a 25 per cent solution of strong sulphuric acid in water. This decolorises everything but acid-fast bacilli which are stained bright red.

Three types of tubercle bacilli are recognised, viz. (1) the human type, (2) the bovine type, and (3) the avian type. This classification is based on differences in virulence and morphological and cultural characters which undoubtedly exist, but it is by no means satisfactorily proved that the types represent different organisms. It is much more probable that the variations are due solely to their environment and to the differences in the soil in which they have grown in successive generations. This view is strongly supported by the fact that bovine infection is comparatively common in man and that avian infection in mammals is also not rare. In other words, the disease, as set up by either of the three types of organism, is intertransmissible, and the lesions produced are fundamentally similar in each case.

The tubercle bacillus may be destroyed by heat, a temperature of boiling-point being fatal to it in one minute, or a temperature of 85° C. for five minutes, or 80° C. for fifteen minutes.

The susceptibility to the disease varies in different animals, depending probably upon the condition of the individual at the time of infection, *i.e.* a healthy robust animal is less likely to contract the disease than a poorly nourished animal in a weak, run-down condition under similar exposure. Any circumstance which tends to reduce the vitality of a person or animal predisposes to an attack of the disease if the organism is present. On the other hand, given a sufficiently large dose of the organisms the disease will be set up independently of the bodily condition of the animal.

Methods of Infection.—Infection may take place either by inhalation, by ingestion, or by inoculation, either through the skin or into the udder or vagina. It may also be transmitted congenitally.

(1) *By Inhalation.*—In cattle and in the human subject undoubtedly the commonest method of infection is the inhalation of tubercle bacilli suspended in the atmosphere, having been expectorated by persons or animals affected with pulmonary tuberculosis. This is shown by the fact that in cattle and man the primary lesions of tuberculosis are in the majority of cases either in the lungs or thoracic lymphatic glands. Air-born tubercle bacilli may also arise from partially desiccated material discharged from other sources, such as fæces, urine, or discharging tubercular sores.

(2) *By Ingestion.*—This is the commonest method of infection in the horse, pig, and fowl, and probably in the dog and cat, and is due to the consumption of tubercle bacilli with food or water. The source of the bacilli in this case is the same, with the addition of tuberculous milk and offals. The common primary seats of the lesions where infection takes place by this channel are the glands of the throat, the intestinal mucous membrane, and the mesenteric lymphatic glands.

(3) *By Cutaneous or Subcutaneous Inoculation.*—This is exceedingly rare in the case of the lower animals, but the so-called "lupus" of the human subject may be an example and also "bumblefoot" in poultry. The rare cases of infection of the mammary gland through the teat may perhaps be included in this category.

(4) *By Way of the Urino-genital Tract.*—This is a somewhat rare method of transmission. A tubercular bull, however, may transmit the disease during service, and it is also possible that a healthy bull may transfer the disease to a healthy cow after service of a cow affected with tubercular metritis. The bull may similarly contract the disease himself.

(5) *Congenitally.*—This is also an extremely rare method of infection, and it is doubtful if more than one tubercular cow in a thousand gives birth to a calf already infected. Whenever it does occur infection takes place from a tubercular placenta, the mother being the subject of a tubercular metritis. In this case the liver of the newly-born animal is the constant seat of lesions. It is practically certain that the male parent never transmits congenital tuberculosis.

Distribution of Tubercle Bacilli throughout the Body.—When tubercle bacilli enter the body they may pass into a mucous membrane or other tissues and become destroyed by the natural powers of resistance. If they are not, they immediately multiply and produce a local inflammatory lesion, which, owing to its nodular character, is usually called a *tubercle.* They then enter the lymph stream and are carried to the lymphatic glands associated with the seat of entrance, and there set up a similar tubercle to the primary lesion. From these glands they may pass on to other lymphatic glands or to the blood stream. Sometimes they gain direct access to the blood stream from the primary lesion, owing to the implication of the wall of a blood-vessel in the lesion. At other times the lesion may extend and involve adjacent structures, setting up new tubercles, which in turn may be responsible for other sets of lymphatic glands becoming involved. The rapidity of spread of these lesions depends largely upon the virulence of the particular strain of bacilli, and also on the powers of resistance of the individual.

When inhalation is the channel of infection, the primary lesion usually occurs in the lung tissue, from which the bronchial and mediastinal glands become involved. The lesion may also extend from these tissues to the pleura, setting up a serous tuberculosis or tubercular pleurisy, from which the lymphatic glands of the chest wall may become affected. From the pleura and thoracic glands bacilli may invade the pericardium and heart muscle, setting up tubercular pericarditis and myocarditis. Infective material may also pass through the diaphragm and in this way set up a tubercular peritonitis. From the primary lesion in the lung, organisms may be coughed up into the pharynx and set up a tuberculosis in the region of the throat and the pharyngeal lymphatic glands, while other organisms being swallowed from this situation may give rise to abdominal tuberculosis in the same way as if the original method of infection was by ingestion.

When the mode of infection is ingestion, a primary lesion may occur in the pharyngeal lymphatic glands and also in the abdominal cavity. When tubercle bacilli are swallowed, they may set up lesions in the intestines, most frequently in the form of ulceration of the ileum, and from this seat the organisms invade the mesenteric lymphatic glands. From these glands organisms may gain the blood stream, as in the case of pulmonary tuberculosis, and set up generalised tuberculosis, or they may extend to the peritoneum, causing tubercular peritonitis. From the peritoneum the uterus may become infected, and most cases of mammary tuberculosis are also secondary to peritoneal tuberculosis. In each case, however, infection may occur by way of the blood stream or directly by the introduction of organisms during coition or through the teat respectively.

When tubercle bacilli are introduced into the skin and subcutaneous tissues a local lesion may develop with secondary lesions in the corresponding lymphatic glands, from which extension may take place as already indicated.

It will thus be seen that the main channel of spread of tubercle bacilli throughout the body is the lymph stream, usually but not invariably in the normal direction of flow of lymph. When the organisms, however, gain access to the blood stream they set up, as already stated, a condition known as *generalised tuberculosis*. It most frequently happens that it is the venous blood that is invaded, as a consequence of which the chief evidence of generalisation is encountered in the lungs and consists of a large number of very small lesions called miliary tubercles evenly distributed throughout both lungs.

If the blood stream becomes invaded from a vessel wall in the abdominal area, the subsequent lesions will be developed first in the liver, from which the general blood stream may be subsequently invaded.

When the arterial blood is invaded the lesions may be very widely disseminated, and may be met with in any or almost all parts of the body, such as the spleen, kidneys, and other viscera, the nervous system (brain and spinal cord and their membranes), bones and joints, and also in the lymphatic glands embedded between the muscles.

In the rare cases of congenital tuberculosis the primary lesions are also found in the liver owing to the infected placental blood being carried direct to that organ. Subsequently lesions may also be produced in other parts of the body. Congenital tuberculosis is a form of generalised tuberculosis.

Anatomical Changes.—The lesion produced by the tubercle bacillus is first a nodule or tubercle, which gradually increases in size. Comparatively soon the centre of the lesion undergoes necrosis, speedily followed by caseation, *i.e.* formation of yellow substance with the consistence of cheese. In some animals the lesions further undergo calcification, that is, lime salts become deposited in them, giving them a gritty mortar-like consistence (common in pigs). At other times they undergo a softening and produce what is known as caseo-pus (common in horses). These characteristic lesions are rarely or never found in the organs or tissues of the body without the corresponding lymphatic glands being also affected.

In the various tissues tubercles may vary in size from that of a millet seed up to the size of a large orange, and sometimes very extensive areas of organs such as the lungs and liver may be affected, and rendered solid and almost entirely functionless.

In serous tuberculosis the lesions usually consist of clusters of tubercles at first red and soft but later becoming firm and cheesy, sometimes pedunculated. These lesions are generally known to butchers as "grapes."

In the spleen the lesions usually consist of discrete tubercles, more or less numerous, and standing up above the surrounding tissues.

When the udder is affected tubercular mastitis is set up, and the lesion is usually in the form of a diffuse induration. One or more quarters may be involved.

The principal conditions which must be distinguished from tubercular lesions are as follows:

In cattle, echinococcus cysts in the lungs and liver, bacterial necrosis of the liver, and actinomycosis ; in the horse, glanders nodules, and echinococcus cysts in the lungs and liver, and lymphadenoma of the spleen ; in sheep, worm nodules (pseudo-tuberculosis) in the lungs and Hodgkin's disease (lymphadenitis) ; in swine, echinococcus lesions.

In each case differentiation is assured by the distribution and character of the lesions, and the discovery of the tubercle bacillus.

Tuberculosis in Cattle.—All forms of tuberculosis may be met with in cattle, but pulmonary tuberculosis is the predominant form. It usually runs a chronic or slowly progressive course, being slow and insidious in its onset. It is occasionally acute, the acute form being a sequel to the chronic. In the early stages there are no appreciable symptoms, and the general condition of the animal may afford no information, fat and thin cattle being equally affected. Later, a short intermittent cough is set up, becoming soft and moist, more or less chronic, and always increased on exercise. The animal then begins to lose condition and slight nasal discharge, usually muco-purulent, is set up, in which tubercle bacilli are occasionally found. When the cough becomes very bad a quantity of bronchial secretion is expelled by the nose and mouth, and the breathing becomes frequent and shallow. The temperature is no guide whatever, but if taken at frequent intervals will usually be found to fluctuate considerably. The mucous membranes are only affected in advanced cases when they become anæmic. On auscultation increased bronchial sounds may be observed in certain areas, together with loss of respiratory sounds in the consolidated areas. When the pleura becomes invaded a slow, insidious, painless pleurisy is set up. Friction sounds may be observed on auscultation, but there is very little tendency to accumulation of the fluid in the chest. In some cases the bronchial lymphatic glands may become very large and interfere mechanically with respiration by pressure on the trachea or bronchi, while pressure may also be exerted on the œsophagus and set up hoven or tympanites.

In *acute* pulmonary tuberculosis exaggerated respiratory symptoms are developed resembling those of acute pneumonia. They follow on gradual wasting and the usual symptoms of chronic pulmonary tuberculosis. The animal becomes suddenly very distressed with blowing respiration, mucous membranes injected, the temperature 105° or 106° F., very rapid pulse, and great loss of flesh. On auscultation there are considerable alterations in respiratory sounds, which are either increased in the early stages or lost in the later stages. In such cases death usually occurs within a week and is preceded by general collapse.

Acute miliary tuberculosis of the lungs may arise secondarily from abdominal tuberculosis.

Abdominal tuberculosis in cattle is often secondary to pulmonary tuberculosis as the result of the patient having coughed up bacilli into the pharynx and swallowed them. With this affection animals show increased emaciation and loss of condition, the mucous membranes becoming very pallid, temperature low and may be subnormal, pendulous abdomen, staring coat, and frequently diarrhœa of a very persistent character. Occasionally enlarged lymphatic glands may be felt on examination per rectum.

Tuberculosis of the liver is comparatively common, but it cannot be definitely diagnosed during life.

The ovaries are often affected at the same time, resulting in recurring œstrum (nymphomania) and sterility.

Uterine tuberculosis is occasionally seen, but is not very common. It is a slow form of metritis, and gives rise to a turbid muco-purulent vaginal discharge, rarely blood-streaked. In the less severe cases pregnancy may occur and the fœtus may be carried the full period of gestation. In the majority of such cases the calf would probably be affected with congenital tuberculosis. As already noted, if the ovaries are affected, frequently recurring œstrum is produced and sterility. The vulva is very rarely involved, but cases have been observed with great thickening resembling elephantiasis.

In the bull, tuberculosis of the testicle is occasionally met with. The inflammation is a cold one, the testicles becoming enlarged, more dense than normal, and painless. It produces sexual excitement and often sterility. The penis is also very rarely affected, exhibiting firm tubercles at its free extremity or an apparently granulating tumour just posterior to the glans penis.

The superficial lymphatic glands may become tuberculous, with the production of large tumour-like growths, which may attain the size of a coco-nut. The common situations of these are the region of the throat, the base of the neck, the flank and above the mammary gland. In the neck and throat they are often called "wens" or "clyers." They are more or less firmly attached to the surrounding tissues, and are firm and dense on manipulation. Sometimes they ulcerate, and burst and discharge thick creamy pus, slightly blood-tinged. In the throat they may interfere with respiration and with swallowing.

Tubercular mammitis or mastitis is set up when the udder becomes affected. It takes the form of a diffuse rather slowly progressive induration. It usually commences in one quarter, but may eventually involve the whole udder. The affected quarter or quarters gradually increase in size and density, and in rare cases the udder may actually weigh as much as 40 or 50 lbs. The disease appears to be comparatively painless. In the writer's experience the right hind-quarter is most frequently involved, and this view is supported by a record of 100 consecutive cases by Mr. J. S. Lloyd, which he summed up as follows: "The right hind-quarter only was affected in 38 cases, the left hind-quarter in 29 cases, both hind-quarters in 10 cases. The right fore-quarter only was affected in one case, whilst the right half (fore- and hind-quarters) of the udder was affected in seven cases. The left fore-quarter only was affected in four cases, the left half of the udder in four cases, and all four quarters in four cases. Both fore-quarters were affected in one case, the right-hind and left-fore in one case, the right-hind and both fore-quarters in one case."

This record shows that the right hind-quarter was involved in 61 per cent of cases, the left hind-quarter in 47 per cent of cases, the right fore-quarter in 14 per cent of cases, and the left fore-quarter in 15 per cent of cases. The possible explanation of this may be found in the fact that cattle most frequently lie on their right side to facilitate rumination as compared with lying on the left side, and as the infection is usually a backward extension from the supra-mammary lymphatic gland, the right hind-quarter would thus be the first to be affected.

The milk at first is usually unaltered in appearance. It may be slightly increased in quantity. Later it appears thin and watery, and in advanced cases the secretion becomes much diminished, and the glands may become almost functionless. The milk is occasionally but very rarely blood-tinged. Tubercle bacilli are always present in the milk of tubercular glands, though in the earlier stages it may be difficult to demonstrate them microscopically. In suspected cases the milk should be treated with antiformin and centrifuged, the sediment being examined microscopically. In the early stages it may not be possible to find the organisms in this way, and the suspected material has then to be injected into guinea-pigs. The guinea-pigs may be killed after a month, and if the material contains tubercle bacilli, tuberculosis will be found. Tuberculous milk can be rendered quite harmless by boiling for one minute, or raising the temperature for a few minutes to 85° C.

Tuberculosis of the brain and meninges may occur in adult cows and also in calves, in each case due to generalization. The symptoms set up are extremely varied. Where the meninges alone are affected there is great excitement, restlessness and sometimes delirium or frenzy, while in the advanced stages and where the brain substance is affected the symptoms are rather more depressant in character. An animal may push his head forward in a corner or may become partially paralysed; great depression and coma supervene, followed by death after a few days.

Tuberculosis of the eye may also be met with, but is rare. When it does occur it is most often due to generalization. It may affect the conjunctiva, the cornea, the retina, or the interior of the eye-ball, and cause blindness.

Tuberculosis of the skin may occur in the form of a string of farcy-like buds the size of a walnut or larger. The writer has seen several such cases in feeding-bullocks of two years old. Exploratory punctures of the lesions were made, and in the thick creamy pus obtained tubercle bacilli were readily demonstrated.

Tuberculosis of the Horse.—The abdominal form of tuberculosis is most commonly met with in horses, the mesenteric glands and the spleen being the chief seats of lesions.

The *symptoms* are general unthriftiness with a capricious appetite and pallid mucous membranes. The abdomen is usually pendulous, but occasionally tucked up. The temperature is no guide, and is often subnormal. There is excessive urination, and in the advanced stages constipation, which is followed by a persistent diarrhœa with rapid wasting and progressive debility. On examination per rectum the enlarged lymphatic glands may be felt in the roof of the abdomen in the region of the kidneys, and where the spleen is involved that may sometimes be similarly felt. The vertebræ of the neck are not infrequently affected and enlarged in the horse, producing a stiff neck with or without a curvature.

Pulmonary tuberculosis may also be met with. In rare cases the lungs may be affected primarily with a few lesions, causing a short weak cough and general loss of condition. The pulmonary lesion, however, is far more commonly an acute miliary tuberculosis of the lungs, secondary to the abdominal disease. When this occurs the symptoms are those of acute pneumonia with a high temperature, blowing, difficult respiration, rapidly progressive weakness and death after a few days.

Tuberculosis in Pigs.—In swine, tuberculosis is most frequently met with in dairying districts, the main vehicle of infection being milk or fæces of tubercular cows. Young pigs are more often affected than adults. The abdominal organs are therefore mainly affected, producing gradual wasting, anæmia, pallid skin and often diarrhœa. The throat is frequently swollen owing to lesions of the pharyngeal lymphatic glands. Generalization frequently occurs in pigs, and lesions are rapidly produced in other parts of the body, including the lungs and spleen.

When generalization occurs emaciation is extremely rapid, and the animal soon dies in a state of cachexia. A primary pulmonary tuberculosis is occasionally met with in adult pigs, giving rise to a cough and blowing respiration, occasional vomiting, anæmia, emaciation, and death. The bones, and particularly the vertebræ, are more frequently tuberculous in the pig than in any other animal, but tuberculous ostitis can rarely be diagnosed during life.

Tuberculosis of the Dog and Cat.—In these animals both the abdominal and pulmonary forms are comparatively common, and give rise to great emaciation and progressive anæmia. The appetite is variable and the temperature is no guide.

In the pulmonary form there is difficult respiration, rapid and shallow, but frequently no coughing. Sneezing occasionally occurs, especially in cats, and in such cases the nasal discharge may be rich in tubercle bacilli. On auscultation one may find excessive bronchial sounds, and in cats sometimes phthisical cavities due to the breaking down of lung lesions. Sometimes there is very rapid exudation into the chest, causing intense dyspnœa.

In the abdominal form in the dog, dropsy of the abdomen is frequently produced. The mesenteric glands may be felt enlarged on palpation through the abdominal wall, and even more frequently the considerably enlarged liver may be felt through the abdominal wall behind the ribs, particularly on the right side.

In the abdominal form in cats, dropsy may be met with, but not so frequently as in the dog. The enlarged mesenteric lymphatic glands can be more constantly recognized, especially those towards the terminal part of the ileum and about the cæcum. They can usually be readily distinguished in the posterior third of the abdomen, and vary in size from half a hazel nut to a tangerine orange.

In both dogs and cats the muscular wasting is very pronounced, and perhaps this is especially the case in the temporal muscles. At the same time the eyes become deeply sunk into the head and may be affected with catarrhal conjunctivitis. In both species the pharyngeal lymphatic glands may also be affected. They are enlarged, dense and almost painless, and are more or less firmly adherent to the surrounding structures. By this latter fact they are readily distinguished from enlarged thyroids.

Tuberculosis of the eye may also be met with, the retina being frequently involved, causing blindness without any gross alteration in the eyeball. The pupils are usually widely dilated.

Diagnosis of Tuberculosis.—In the early stages of the disease it is difficult or almost impossible to make a positive diagnosis from the symptoms exhibited. Even in some more advanced cases the symptoms may closely resemble those of other wasting diseases, thus necessitating other measures than the observation of symptoms.

The presence of tubercle bacilli may be demonstrated in the tissues or some of the discharges by microscopic, cultural or experimental methods, but these methods are not always available. In the case of the death of an animal the post-mortem examination is the most certain method of positive diagnosis. During life it is frequently necessary to resort to the use of a diagnostic agent, viz. tuberculin, which is a sterile glycerinated solution of the toxins of the tubercle bacillus cultivated in broth and containing 0·5 per cent carbolic acid as a preservative. This is the so-called "Old Tuberculin."

There are several methods of employing tuberculin for the diagnosis of tuberculosis as follow:

(1) *The Subcutaneous Tuberculin Test.*—This is the method most commonly employed. The animal to be tested must be kept indoors while under the test, and the temperature should be taken several times before the inoculation in order to ensure that the suspect is not already in a fevered condition. The tuberculin may be injected anywhere under the skin, preferably where there is a fair amount of loose subcutaneous tissue. Probably the best site of injection in cattle is just in front of the shoulder; in horses the side of the neck; in dogs under the skin of the back. The dose of tuberculin depends largely upon the degree of concentration. In English prepared tuberculin the dose for adult cattle is 3 c.c., for large bulls 5 c.c., for horses 8 c.c., for dogs about ½ c.c., and for cats ¼ to ⅛ c.c. It appears to be quite unreliable for swine. A sterile hypodermic syringe must be used, and the site must be disinfected before the operation. After injection the temperature should be taken—in horses and cattle at the ninth, twelfth, fifteenth and eighteenth hours. In some cases a reaction sets in as early as the sixth hour. If there is any reason, however, to suspect that the animal has been tested a short time previously, a double dose should be given and the temperature should be taken at the third and sixth hours after injection. In the case of dogs and cats the temperature should be taken at the second hour after injection and each hour up to the eighteenth, since in these animals a much earlier reaction is frequently provoked.

Animals affected with tuberculosis react in the form of a rise of temperature, which in a typical case is gradual from about the eighth to the sixteenth or eighteenth hour, after which it gradually subsides and returns to the normal in from twenty-four to thirty-six hours. In some cases the temperature rises to 105°-106° F. and may remain high for twenty-four hours. The extent of the temperature reaction does not, however, bear any direct relationship to the degree of the infection, and it often happens that the higher degree of temperature is registered in the mildest cases. A rise of about 2·5° F. above the normal for the individual (from 101·5° to 104° F. in cattle) is a positive reaction, and the animal must be regarded as tuberculous. If the temperature remains below 103° the animal is not tuberculous, while if the temperature is between 103° and 104° it should be regarded as a doubtful reaction, and the animal should be re-tested in about a month. During reaction the patient is often somewhat depressed and off its food. The pulse and respiration are accelerated, and in the case of milch cattle the milk yield is often diminished and may remain a little below the average for a day or two. The application of the test does not in any way interfere with the usefulness of the milk for food, that is to say, that if the milk was previously safe for human consumption it is still fit to be used. The vascularity of lesions during a reaction may be temporarily increased, so that superficial tubercular glands may be felt somewhat swollen and hot. During the application of the test the animal must be kept in an even temperature if possible, and there should not be any immediate change of diet. Any elevation of temperature more than 1° or 2° above the normal renders the test inapplicable, and it is not reliable in the case of animals in a very advanced stage of the disease. Tuberculin would also fail to produce a reaction in the incubative stage of tuberculosis. The suspect must not have any drugs administered before or during the test.

Tuberculin in doses used for diagnostic purposes has no effect whatever upon healthy animals, while on the other hand it reveals the presence of tuberculosis even in the smallest areas. A positive reaction to a tuberculin test may be regarded as a certain evidence that the animal is affected with tuberculosis. It may happen, however, that on post-mortem examination of reactors no macroscopic lesions may be discovered. That should be regarded as evidence of the searching effect of tuberculin, which reveals the presence of some small hidden focus of the disease rather than as an indication of fallibility.

The subcutaneous tuberculin test is extremely reliable in cattle, and almost as reliable in horses. Although of considerable service it is not so reliable in the smaller animals.

(2) *The Ophthalmic or Conjunctival Tuberculin Test* (*Calmette's*).—This test is carried out with a specially prepared concentrated tuberculin containing no carbolic acid. In applying the test the animal's head must be held to one side with one eye pointing upwards. A few drops (not less than five or six) of tuberculin are dropped into the conjunctival sac. The head should be held in this position for a little while until the tuberculin becomes distributed under the lids. The other eye is left as a control, and the two eyes are examined at intervals from the eighth or ninth hour after instillation of the tuberculin.

If the animal is tuberculous there is a reaction in the tested eye consisting of a temporary conjunctivitis with the collection of a purulent secretion at the inner canthus of the eye. The reaction may last for twenty-four hours or several days. At the same time the other eye remains healthy and is not affected with catarrh. The severity of the reaction is no indication of the extent of the disease.

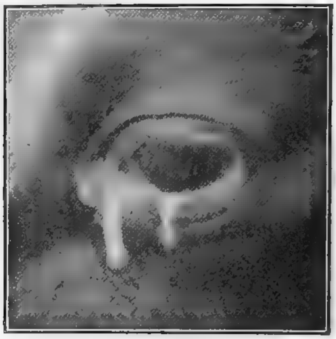

Fig. 12.—A positive reaction to the conjunctival tuberculin test. (From the *Journal of Comparative Pathology and Therapeutics.*)

The advantages of the conjunctival test are that it can be applied irrespective of the temperature of the suspect, and, moreover, one application of the test does not render a subsequent application useless—in fact it may produce a more pronounced reaction; the test may be repeated after four or five days, and the previous application of the subcutaneous test does not interfere with the conjunctival test.

Under these circumstances the ophthalmic test will be found very useful, and in the case of a positive reaction the suspect may be definitely regarded as tuberculous. On the other hand, however, it is not quite so reliable as the subcutaneous, since tubercular animals sometimes fail to give a positive reaction.

(3) *Combined Ophthalmic and Subcutaneous Tests.*—The subcutaneous and ophthalmic tests are sometimes carried out simultaneously. This procedure is probably better than either test carried out alone, especially as the ophthalmic test may provoke a reaction in an animal previously injected with tuberculin subcutaneously.

(4) *The Intradermic Test.*—This test consists of the injection of 4 or 5 mm. of special concentrated tuberculin into one of the folds of the skin at the base of the tail and at the side of the anus. In order to inject the tuberculin the tail must be raised to render the skin tense. In a positive reaction the fold of skin into which material has been injected becomes

œdematous and swells up to the size of a Brazil nut or larger in about twenty-four hours; the swelling persists for several days. The opposite fold of the skin which has not been injected constitutes a control and undergoes no change.

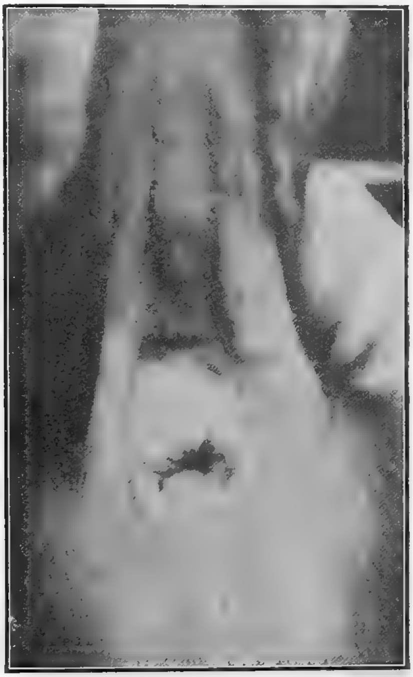

FIG. 13.—A positive reaction to the so-called intradermic tuberculin test. (From McFadyean, *Journal of Comparative Pathology and Therapeutics.*)

(5) *A Cutaneous or Dermic Reaction* (*Pirquet's Reaction*).—In this test a small area of the skin, usually of the shoulder, is shaved and disinfected, and scarified with a sharp knife. It is then painted over with a concentrated tuberculin. In a tubercular animal in a positive reaction the skin becomes œdematous and swollen twenty to thirty hours later, and persists for four or five days. This may be followed by the formation of pustules and a desquamation of epidermis. There is no general or systemic disturbance, and the test may be repeated after an interval of a few days. A previous application of subcutaneous test is said to diminish this reaction.

(6) *The Epidermal Test or Dermo Reaction.*—In this test an area of the skin is shaved and disinfected, and an ointment consisting of concentrated tuberculin and lanolin is rubbed into the part without previous scarification. In a positive reaction the area becomes red, swollen and œdematous, and somewhat painful in about twenty-four hours. There may be also the production of vesicles and pustules.

Treatment.—Tuberculosis is an incurable disease, although all cases of tuberculosis do not prove fatal. There is, however, great risk of the disease being conveyed to other animals or to the human being, and consequently steps should certainly be taken to control the disease.

General preventive measures can be adopted by immediately isolating any animals that have reacted to the tuberculin test, and practising thorough disinfection of the shed or sheds in which they have been kept, in order to reduce the danger of spread. Cattle should be kept outdoors as far as possible, and when it is necessary for them to be housed, the best hygienic conditions of the sheds should be provided, with special attention to light, ventilation, and an air space of not less than 800 to 1000 cubic feet per cow.

In the campaign against bovine tuberculosis two methods have been adopted with a certain amount of success, viz. Bang's method, which is a method of eradication, and Ostertag's method, which aims at control rather than eradication. There is only one method by which tuberculosis can be eradicated from any herd, and that is by the use of tuberculin for the diagnosis of the disease and the isolation of all reactors, in order to prevent infection spreading from them to the healthy animals.

Bang's Method.—All the animals of the herd are first carefully examined and the obviously affected cattle are condemned and slaughtered, either at once or after a time to fatten them for the butcher, their fitness for food being determined on examination after slaughter. The remaining cattle are then submitted to the tuberculin test, and the reactors are separated from the non-reactors and placed in a separate building. No new cattle must be admitted into the herd unless previously tested and proved free of the disease. The healthy herd must be re-tested at intervals of not more than a year, preferably six months or even less, and any that have developed the disease in the interval must be relegated to the infected herd. Only perfectly healthy bulls should be used. The calves of the healthy herd may remain with their mothers or be provided with pasteurized milk. The calves of the reactors should be separated from their dams immediately after birth, and fed on pasteurized milk or other tubercle-free foods. All the calves should be tested with tuberculin at six months old and again at ten or twelve months old, and if then free from tuberculosis they may be admitted to the tubercle-free herd. The reacting cows may be used for breeding so long as they show no clinical evidence of the disease. If, however, they develop symptoms of tuberculosis they should be slaughtered. In this way a tubercle-free herd may be ultimately built up.

Ostertag's Method of Control.—In this method of control of tuberculosis reliance is mainly placed on clinical evidence and not on the use of tuberculin. The cattle are examined, and all those affected with so-called "open" tuberculosis are immediately removed from the herd. Those not in calf are slaughtered, while the in-calf cows are allowed to give birth to their calves, which are immediately removed from their dams. The cows are then slaughtered. By "open" tuberculosis is meant those cases in which the tubercle bacilli are escaping from the body, and which are therefore dangerously infective to animals and man. The opposing term "closed" tuberculosis implies that though the animals may be infected they are not discharging tubercle bacilli, and animals so affected are regarded for the purposes of this method as being not dangerous. The cows retained in the herd are examined about every six months, and all suspicious cows are removed and slaughtered. The examination includes the bacteriological examination of milk and any other excretion which may be suspected of containing tubercle bacilli. All calves must be reared on pasteurized milk, or on new milk from cows known to be free from tuberculosis.

The terms "open" and "closed" tuberculosis are not to be recommended, since animals affected with the latter condition, which implies a certain degree of safety, may at almost any time, without warning, commence to discharge tubercle bacilli by various channels. Such cows, therefore, become seriously dangerous, and may spread infection throughout the herd for a considerable time before being discovered. Consequently, although Ostertag's method clears away the most dangerous animals fairly rapidly, and may assist in the reduction of tuberculosis, it will not result in a tubercle-free herd, and on that ground compares unfavourably with Bang's method.

No effort to eradicate or even to adequately control this disease is likely to be satisfactory without resort to the use of tuberculin. The tuberculin test should be applied at least every six months, and the reactors removed as suggested by Bang. The entire herd should in addition be carefully examined at least every two or three months by a veterinary surgeon. With a view to encouraging farmers to adopt some satisfactory mode of eradicating the disease, Hoare suggests that a capitation grant should be allowed to owners for a limited number of years for each animal found to pass the tuberculin test. This suggestion has much to commend it, since it would go far to compensate the owner for the considerable expense entailed by any real effort to build up a tubercle-free herd.

With regard to the international traffic in pedigree cattle, it is obviously necessary that only tubercle-free cattle should be allowed to be introduced for purposes of improving the breeds. With this end in view reliance can only be placed upon the use of tuberculin, which, as already pointed out, has certain limitations, the most important of which are (1) that it may fail to provoke a reaction in animals seriously affected with the disease, and (2) that it will not provoke a reaction in cattle in the incubative stage or that may have been recently injected. The first stated objection is not of much importance, since advanced cases of the disease are readily detected by clinical methods and are not likely to be purchased for export. In order to overcome the second objection a period of quarantine and isolation should be enforced before the test is applied. The effects of a previous injection of tuberculin would by this time have passed away, while animals in the incubative stage would in all probability have developed such a lesion as would enable tuberculin to provoke a reaction. This procedure should be carried out before the animal is shipped to a foreign country, in order to avoid disappointment on the arrival of the animal by its subsequent reaction to the test.

In order to encourage this method so far as the export of British cattle is concerned, the Government has instituted a testing station providing quarantine and isolation before submitting the cattle to the test. Cattle receiving a clean bill of health under these circumstances are likely to prove non-tuberculous on reaching their destination.

The prevention of tuberculosis in other species than cattle depends very largely upon the prevention of consumption of contaminated food materials, such as milk of tuberculous cows, milk offals and the flesh or viscera of tuberculous animals unless previously sterilized. Consequently all such materials should invariably be cooked before being fed to swine and also to dogs and cats. Swine should not be allowed access to the fæces of cattle. Moreover, infection from diseased animals of the same species must be prevented by the slaughter of all affected, diagnosis being effected either by the observation of clinical symptoms or the use of tuberculin.

Immunization.—It has been abundantly shown that increased resistance to natural or artificial infection with tuberculosis may be produced by various methods, but none of these methods at present afford such results as to justify their general adoption.

The best results have been obtained by injecting calves intravenously with cultures of tubercle bacilli of the human type, and this is sometimes followed by a second inoculation several months later. The immunity thus resulting is apparently of short duration, and the injected bacilli are not readily absorbed and may exist in the body for some considerable time. On account of this fact Vallée considers that the animals are rather in a condition of latent infection. It is asserted that heifer calves thus treated may discharge tubercle bacilli years later with their milk. This is obviously a very serious objection to the procedure, at any rate in the case of female animals, and cannot therefore be recommended. McFadyean, however, is of opinion that " in valuable infected pedigree herds in which it is considered indispensable to allow the cows to suckle their own calves, some benefit may be derived from the vaccination of the bull calves immediately after birth " ; and in his experience " the operation lessens the chance of infection during the period of suckling as estimated by the results of tuberculin tests after operation. On the other hand, in a considerable number of cases presumably healthy young cattle reacted for long periods after vaccination."

Experiments have also been carried out with vaccines prepared with bacilli of the avian type. The degree of resistance afforded by this means is, however, much less than when human tubercle bacilli are used.

Other methods of vaccinating with dead or avirulent cultures have not produced any serviceable immunity against infection with bovine tubercle bacilli.

Human and Bovine Tuberculosis.—It is not possible here to enter into a discussion as to the identity or otherwise of tubercle bacilli of the human and of the bovine types. It is of far greater importance to emphasize the fact that although the most common source of tuberculosis in the human being is the person affected with tuberculosis of the lungs, who is frequently and in some cases constantly expelling tubercle bacilli, yet a considerable number of human beings are undoubtedly affected with tuberculosis due to bacilli of the bovine type. This is particularly the case with young children fed entirely or mainly on cow's milk. Such milk may become infected with tubercle bacilli either as the result of tuberculosis of the mammary gland or as the result of contamination with tubercle bacilli from expectorate, fæces, or other discharges. In view of the fact that probably not less than 30 per cent of the dairy cows of this country are affected with some form of tuberculosis, there is little prospect that the disease will be eliminated in the near future, and it is therefore highly improbable that a tubercle-free milk supply can be guaranteed. Consequently, the only real safeguard for children consists in pasteurizing the milk, that is, raising it to a temperature of 85° C. for five minutes. The nutritive value of such milk is not in any way reduced.

The danger to adults is less than to infants on account of the fact that milk constitutes a far smaller proportion of the total food consumed, nevertheless cooked or pasteurized milk should be recommended, unless the herd from which the milk is obtained is known to be tubercle free.

The danger from the consumption of the flesh of tuberculous animals is far less than from milk, particularly as it is rarely taken in an uncooked condition. There is great danger, however, in the traffic in tuberculous meat, owing to the ease of contamination of other articles of diet during the process of handling.

One should also realize the danger of the presence of tuberculous dogs and cats, each of which may be affected with either pulmonary or abdominal tuberculosis and may be discharging considerable quantities of tubercle bacilli, in the majority of cases of the bovine type, but sometimes of the human type.

G. H. W.

JOHNE'S DISEASE

Chronic Bacterial Enteritis of Cattle ; Chronic Paratuberculous Enteritis of Cattle

Johne's Disease is a chronic enteritis particularly affecting cattle, and having a very wide distribution throughout the world. In addition to cattle, sheep and goats may be affected, and several cases have been seen in horses, and one in a deer.

Etiology.—Johne's disease is due to a small acid-fast bacillus in some respects resembling the tubercle bacillus. It has been called the " Bacillus of Johne's Disease," and also " The Paratubercle bacillus." That the organism is not identical with the tubercle bacillus is proved by the fact that it is non-pathogenic for rabbits and guinea-pigs; and, moreover, Johne's disease is common in the Channel Islands, where bovine tuberculosis is non-existent.

Infection takes place by way of the mouth through the medium of foods which have been contaminated by excreta of infected animals, since bacilli are voided in large numbers with the fæces, thus rendering pastures contaminated. For this reason also isolated cases are unlikely to occur, and there is usually in any outbreak a history of the previous existence of cattle affected with a wasting disease. The disease is very slow of development, and months and even years may pass between the time of infection and the development of symptoms. In other cases infected animals may never show any symptoms of the disease at all.

Symptoms.—The first symptom observed is loss of condition and general unthriftiness, in spite of a good appetite and a plentiful supply of food. The coat becomes harsh and staring, and progressive emaciation sets in. In milch cows and milch goats there is a diminution in the milk yield. The affected animal may remain in this state for a considerable time, fluctuating in condition, with occasional relaxation of the bowels. The temperature varies, being sometimes normal, sometimes elevated several degrees. Eventually diarrhœa sets in which is profuse and persistent, often fœtid, the excreta frequently containing bubbles.

Parturition has a very marked effect, and cows which may have previously been unsuspected of being affected, suddenly after calving develop the disease in a more or less acute form with profuse diarrhœa, waste very rapidly and die in a few weeks. In cases where symptoms are well marked recovery is exceedingly rare, and although feeding and rumination may continue capriciously up to the end the patient ultimately dies of anæmia and exhaustion.

In goats and in sheep the symptoms correspond very closely with those of cattle affected, except that the diarrhœa is rarely so pronounced, the predominant symptoms being emaciation, progressive anæmia, and intermittent diarrhœa.

In the horse similar symptoms are also shown, viz. progressive emaciation and diarrhœa, and death from exhaustion.

Diagnosis.—This affection is frequently included in the general description of " Wasters " or " Piners " ; the other conditions included in that term being tuberculosis and parasitic affections of the alimentary tract. A positive diagnosis, however, can sometimes be made by microscopic examination of a small particle of mucous membrane pinched off per rectum, or sometimes by the examination of fæces previously treated with antiformin and stained by the Ziehl-Nielsen method, the organisms being found in groups or clusters. It is not, however, always easy to discover the organisms in affected animals, and failure to demonstrate them does not justify a negative diagnosis.

A diagnostic agent resembling tuberculin has been prepared from the specific bacilli, and has been variously named " Johnin " and " paratuberculin." This agent is used in the same way as tuberculin, and in a number of affected animals produces the positive temperature reaction, but it fails in a considerable number of cases, and is therefore not a very reliable agent.

Better results are obtained by the use of tuberculin prepared from the avian tubercle bacillus, and corresponding exactly with the old tuberculin test for tuberculosis ; the dose is c.c. vi. injected hypodermically. Affected animals give a thermal reaction of from 2° to 4° F. between the ninth and fifteenth hours after injection, the temperature usually falling gradually to the normal by the twentieth or twenty-fourth hour. It is advisable first to submit the suspect to the ordinary bovine tuberculin test a week or so before applying the avian tuberculin test, since tuberculous animals may react to the latter. The converse, however, does not hold, so that if a suspect gives a negative result to the bovine tuberculin test, and subsequently a positive reaction to the avian tuberculin test, the animal may be regarded as affected with Johne's disease. The test is not applicable in the case of animals with a febrile temperature. In such cases the conjunctival test may be resorted to, or the intradermic test in each case with avian tuberculin, and carried out in the same way as for tuberculosis. The two latter tests are, however, much inferior to the subcutaneous test, as frequently badly affected animals fail to react.

The disease is differentiated from parasitic gastritis or enteritis by negative reaction to avian tuberculin, and the presence of numerous eggs of nematode worms in the fæces.

Prognosis.—As a rule, prognosis is unfavourable. An early diagnosis, where possible, is of the greatest importance, owing to the fact that it is not amenable to treatment, and it becomes a question as to the advisability of slaughter or treatment of the affected animal. It is doubtful if recovery ever occurs, though it is possible there are mild non-fatal cases.

In mild cases taken in hand early the condition may be checked sufficiently to enable the animal to become fattened for slaughter. In cases, however, where diarrhœa is well established, death is usually only a matter of a few weeks.

Anatomical Changes.—The lesions consist of a specific enteritis affecting both small and large bowels. The small intestines are principally affected, and particularly the ileum. In mild cases it may be difficult to observe any obvious change, but in the more severe instances the bowel wall immediately gives the impression of being thicker than normal. On exposing the mucous membrane the latter shows a characteristic wrinkled or corrugated appearance, which is most obvious in the last portion of the ileum. In no portion of the intestine in naturally occurring cases in cattle is congestion a marked feature, though there is often some slight congestion of the large intestine. The thickening of the mucous membrane is also less pronounced in the large than in the small bowel. In an acute case in a deer observed by the writer there was an acute congestion of the jejunum with blood extravasation into the lumen of the bowel.

There is no ulceration of the intestine, although the post-mortem changes may rapidly occur and produce an erosion of the epithelium. The mesenteric lymphatic glands are usually larger than normal, and on being cut into have a juicy œdematous appearance. Microscopic examination of scrapings from the affected bowel and from the mesenteric lymphatic glands reveals the organism in considerable numbers in characteristic clumps.

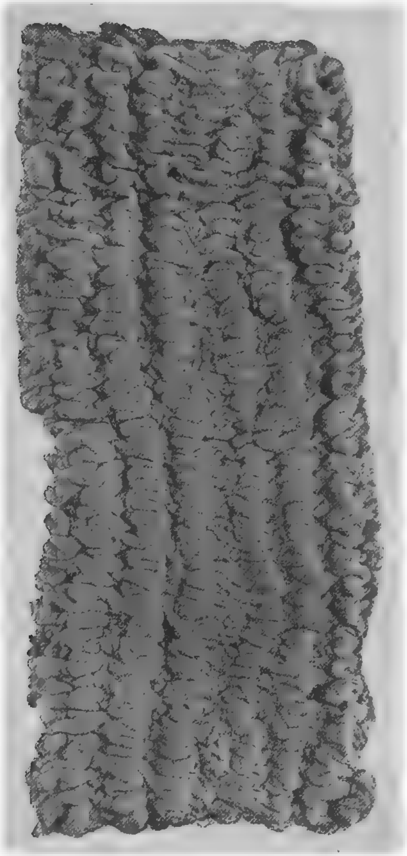

Fig. 14.—Portion of small intestine (ileum) affected with Johne's disease.

Treatment.—If an animal exhibits marked clinical symptoms the best plan is to slaughter at once in order to reduce further infection. Animals that are to be treated should be brought up into the shed or stable and be given a highly nitrogenous diet including corn and cake. Where diarrhœa occurs, decorticated cotton-cake will often assist in checking it.

Medicinally in less severe cases good results may be obtained by the administration of the following mixture suggested by McFadyean:

Ferrous sulphate ℥v.
Dilute sulphuric acid ℥v.
Water sufficient to make 1 pint.

Of this mixture one fluid ounce should be given daily in a pint of water for six or seven weeks.

Other agents that have been employed include salol ʒii. once or twice a day, and cyllin in similar doses. In cases where diarrhœa is pronounced, it may be checked by a combination of opium ʒss, catechu ʒii.-iv., prepared chalk ℥ss.-i., given in flour gruel and repeated twice daily.

In the case of horses, similar doses may be employed as for cattle, while for sheep and goats the same agents may be administered in one-quarter to one-third those doses.

As a matter of prevention all the excreta from affected animals and all soiled litter should either be destroyed or spread on arable land and ploughed in deeply.

On affected premises calves should be reared separately in order to avoid infection. The great difficulty to be overcome in any attempt to exterminate the disease from any herd or locality is the fact that animals may be affected and spread the disease, although not showing clinical symptoms. Reliance must be placed on some diagnostic agent such as avian tuberculin, and the disease systematically attacked as in the case of other contagious diseases. Any attempt to exterminate the disease from a farm must also include attention to sheep and goats, slaughter being probably the best plan of action should any of these animals prove to be infected. The pastures where affected animals have been grazing should be left vacant of ruminants for some time, probably months. It is impossible, however, to state definitely the period required for safety pending further knowledge as to the length of time the organism may persist and remain infective outside the animal body. If practicable, it would no doubt be better that such infected pastures should be put under the plough.

G. H. W.

ACTINOMYCOSIS

"Lumpy Jaw"; "Wooden Tongue"; Ray-Fungus Disease

Actinomycosis is a specific disease most commonly affecting cattle, and characterized by the production of chronic tumour-like growths affecting the connective tissue and bone, frequently with chronic suppurating centres. It is occasionally met with in the horse, pig, sheep and goat, and still more rarely in the dog. The human being is also susceptible.

Etiology.—Actinomycosis is due to a vegetable parasite called the Ray-fungus or *Streptothrix actinomycosis,* which is generally introduced through a wound. It thus belongs to the class Streptothricæ, and is a bacterium, which has the power of branching in its growth. In the lesions it occurs in three forms, viz., clubs, filaments, and spherical forms resembling cocci. The peculiar radiate arrangement of clubs in the colonies of the organism met with in the tissues when seen microscopically is responsible for the name Ray-fungus. The disease has a very wide distribution, being known almost all over the world. It is generally sporadic, occurring in isolated cases, but it not infrequently assumes an enzoötic character, being quite common in some districts and unknown in others. The disease is not contagious in the ordinary sense of the term, since healthy animals may cohabit with affected ones almost with impunity. When it affects several animals in the same herd it is probable that they have contracted it from a common source and not from one another. It appears to be more prevalent in swampy and marshy districts, and it is more commonly met with in barley-growing districts. The frequency with which the disease occurs about the mouth indicates that the parasite is conveyed by food and gains access by wounds or laceration of the mucous membranes. The organism appears to occur commonly as a parasite on various cereals, particularly barley, and on certain coarse grasses, hence it is not surprising to find that barley-grains or pieces of barley-awns or straw have been encountered in the lesions, and have presumably been the means of introducing infection. Slight wounds of the tongue and wounds of the jaw owing to shedding of the teeth afford channels of infection. It may also be introduced through the skin by accidental wounds, and also through operation wounds as after castration. When the mammary gland is affected it is probable that the organism has been introduced through the teat.

The disease is rarely a fatal one and the organisms are limited to the lesions and the discharges from them. The ill-effects are generally produced by mechanical interference with feeding, either by preventing the prehension of food when the tongue is affected, or by interfering with mastication when the jaw-bone is involved, or by interfering with swallowing or respiration. In the majority of cases if the animal is brought under treatment sufficiently early recovery can be obtained or such improvement as will permit it to be profitably fed for the butcher.

Lesions in Cattle.—The common lesion consists of a formation of new fibrous tissue at and around the seat of inoculation of the ray-fungus, frequently with suppurating foci which constitute small abscesses in the substance of the growth. The latter may point and burst, emitting pus which feels gritty on being rubbed between the thumb and fingers. The consistence and colour of the lesion vary. Many are firm and solid, of the consistence of a fibroma, and greyish white in colour. Others have a soft consistence and are yellowish red in colour. On cutting into them one can usually see yellowish specks varying from the size of a small pin-head to that of a pea, scattered through the connective-tissue mass. In those cases with pus production abscesses of varying sizes may be seen, containing pus and surrounded by soft granulation tissue. If a little pus or a scraping from a freshly cut growth be spread on a piece of glass, small granules, yellowish white and about the size of a small pin's head, may be seen. These constitute colonies of the parasite.

When the tongue is affected an indurating inflammation is set up. It commences with the formation of raised nodules which are hard and firm to the touch. Extending inwards from these, there is a marked proliferation of fibrous connective tissue which may extend into the muscular tissue. There is frequently ulceration of the mucous membrane, the ulcers being shallow with sharply defined edges and showing minute yellow spots. The increase in size of the tongue makes that organ too large for the mouth and it is consequently protruded to a varying degree. The tongue lesion rarely suppurates.

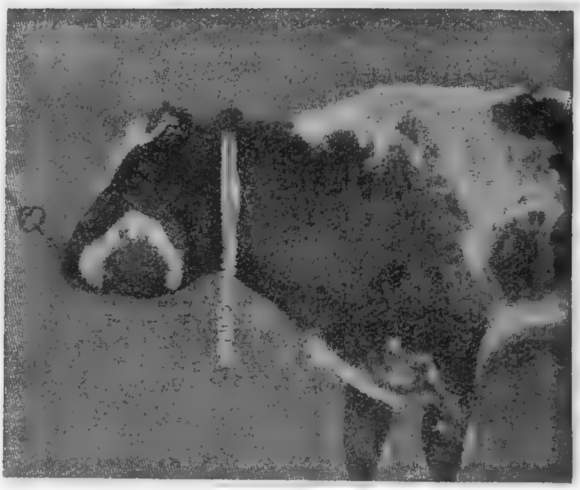

Fig. 15.—Actinomycosis of the jaw.

The outside of the face and the intermaxillary space are affected in a large proportion of cases, but the condition is rendered much worse when the superior or the inferior maxillary bones are invaded. In such cases the circumference of the bone is largely increased, but its density markedly diminished owing to the formation of spongy cavities in it. The skin over the swelling frequently ulcerates and shows granulations. The contained abscesses may burst and discharge the characteristic pus.

Pharyngeal lesions are common. In this situation the growths are usually but not invariably pedunculated, forming polypi which vary in size from a pea to a hen's egg. Sometimes the pharyngeal lesion resembles a simple superficial abscess involving the submucous tissues. In other cases the pharyngeal lymphatic glands become affected and considerably enlarged, and may cause much trouble in diagnosis, since the only discoverable change during life is a more or less constricted pharynx without any surface irregularity.

The respiratory apparatus may become affected with actinomycosis, viz., the larynx and trachea, or the lungs. In the former case the lesions are usually polypoid, but in the lungs the lesions are either tumour-like growths varying in size up to one's fist, containing suppurating foci, or they may be practically abscess cavities. Miliary actinomycosis has also been recorded. The pleura may become implicated by extension from the lungs.

Other rare seats are the mammary glands, the liver, spermatic cord, and stomach. When the mammary gland is affected the lesion closely resembles tubercular mammitis, showing either diffuse induration with small yellow foci or isolated nodules with or without suppuration. Generalization is said to occur, but only very rarely.

Symptoms in Cattle. — The symptoms of actinomycosis almost entirely depend upon mechanical interference and consequently vary with the seat of the lesions.

The lesions take the form of more or less dense fibrous new growths, some being well circumscribed at their bases, while others are diffuse and the line of demarcation between their limits and the surrounding healthy tissues cannot be well made out. When there is a systematic disturbance it is generally due to some complication rather than to the ray-fungus. In uncomplicated cases there is never any elevation of temperature. Respiration, the appetite, secretions, and excretions are not altered unless the growths previously referred to impinge upon the apparatuses concerned. Thus, when the tumour is confined to the skin and subcutaneous tissues frequently no symptoms other than the presence of the growth may be shown. The skin may be either intact, or ulcerated and discharging pus from one or more foci. When the tongue is affected to anything more than a very slight degree prehension is interfered with and may be completely prevented, and the patient loses flesh and may even die of starvation. The tongue is enlarged, protruded, almost as hard as a board, and may be ulcerated. There is dribbling of saliva, and when ulceration is present the discharges are offensive in odour. When the jaw, upper or lower, is the seat of the lesions, in the early stages the inconvenience appears slight, but later mastication is rendered difficult, and so nutrition is not well maintained except with crushed concentrated foods requiring less grinding. The growth here is very likely to burst, and that may occur outwardly or inwardly, the discharge feeling quite gritty. Sometimes the tumour invades the alveoli of the molar teeth which may become loosened or displaced. Not infrequently one finds the skin and muscles of the cheek affected and the bones not interfered with.

The most varying symptoms are shown when the pharynx is affected. If the growth is superficial it is generally pedunculated and may cause intermittent roaring and difficult swallowing. If the polypus passes into the larynx suffocation may occur. If on the other hand it passes during swallowing into the gullet, choking occurs, with its attendant symptoms of tympany, etc. Sometimes a superficial abscess is formed in the pharynx causing difficult swallowing and roaring. If it ruptures the pus may escape by the nostrils or it may be swallowed or pass down the trachea and set up broncho-pneumonia. The disease may invade the glands in the region of the throat and set up roaring and difficult respiration and swallowing, food often being returned by the nostrils. On examination externally one may or may not

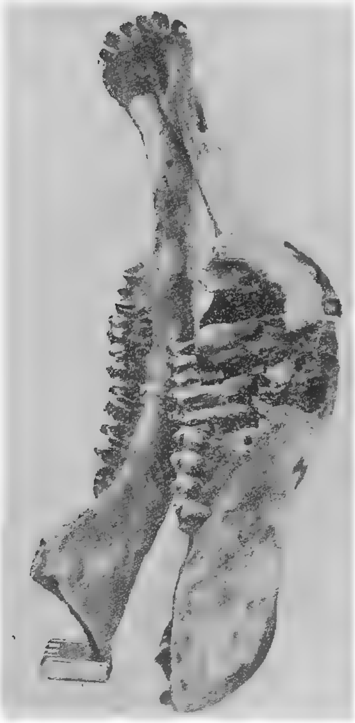

FIG. 16.—Photograph showing actinomycosis as it affects the jaw-bone, causing loose teeth.

appreciate an enlargement in the pharyngeal region, and on examining the inside of the throat with a mouth speculum no irregularity of surface can be felt, and although one may think there is some constriction one cannot be sure since both sides may appear quite the same, and normally the hand would be grasped by the pharyngeal muscles on examining the throat. These cases are generally more acute than the average. The symptoms of dyspnœa may develop rapidly and are continuous, and unless the animal is put under urgent treatment asphyxiation may soon occur. The lips and muzzle are sometimes affected, producing an appearance almost like a hippopotamus, the parts swelling diffusely to such an extent as to occlude the nostrils and cause breathing through the mouth.

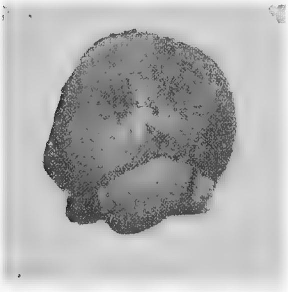

Fig. 17.—Actinomycosis of the nose and tongue.

When the skin and subcutaneous tissues are affected the lesions produced are popularly known as "wens" or "clyers." They may occur anywhere in the skin but are particularly seen about the head and neck. Commencing as a small tumour, a wen may rapidly increase in size up to several times the size of one's fist, sometimes pedunculated, sometimes attached by means of a broad base. The surface of the skin may ulcerate and discharge a purulent material which with brown crusts may cover the growth. In these cases, when the surface is wiped a red granulating area is revealed. The writer has also met with a case in which the fore-leg was affected, becoming diffusely thickened and having a circumference of 18 inches below the knee. Positive diagnosis of this case was only made on post-mortem examination.

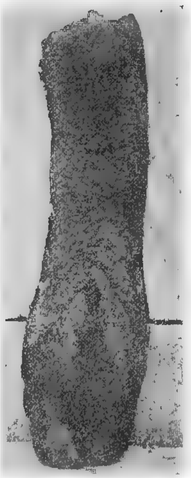

Fig. 18. — Actinomycosis of the leg, causing Elephantiasis.

In the rare cases when the udder is affected the condition is likely to be confused with tuberculosis. The gland may be either diffusely indurated and enlarged, or it may contain a number of firm isolated nodules varying in size from a hazel-nut to a walnut. Positive diagnosis can only be made in such cases by microscopic examination, or by experimental inoculation. A negative reaction to tuberculin may assist diagnosis while a positive reaction would not assist differentiation since the udder lesion may yet be actinomycosis and the reaction provoked by a tubercular lesion elsewhere.

Symptoms in the Horse.—The common seats of actinomycosis in the horse are the submaxillary lymphatic glands, the abdominal wall, and the spermatic cord after castration In the first-named situation the gland gradually increases in size and is very indurated, and may attain the size of a coco-nut. There is a great tendency for it to burst at one or more points and discharge pus in which the colonies of the parasite can easily be demonstrated. Actinomycosis of the tongue and jaw-bones is unknown in the horse. When the spermatic cord is affected a tumour-like mass is produced adherent to the scrotal wall (a form of scirrhous cord) and discharges the characteristic pus from several ulcerating sinuses. A very rare seat of the disease in the horse is the upper lip, which may become considerably thickened and discharge the characteristic pus through small points, both on the surface of the skin and of the inner aspect of the lip. The pus yields the characteristic colonies of the ray-fungus. Actinomycosis of the rectum of a mare has also been recorded.

Symptoms in the Pig.—Actinomycosis of swine particularly affects the mammary glands, producing tumour-like masses possessing a suppurating centre which discharges the characteristic pus. It has also been met with in the throat and in the spermatic cord after castration. Generalization may occur in the pig as in the case of other animals.

Symptoms in the Sheep and Goat.—In these animals actinomycosis is very rare, but it has been described affecting the tongue and lips and jaws, setting up symptoms corresponding with the disease in similar situations in cattle.

In the Dog and Cat streptothrix affections have been met with which are indistinguishable from actinomycosis. In the former the disease may affect the jaw and the mammary glands and the interior of the chest, while in the cat it has been met with in the lungs and in the peritoneum.

Differential Diagnosis.—**In the ox** actinomycosis must be distinguished from *tuberculosis, actinobacillosis,* and from *malignant tumours.* In cases where there is any discharge microscopic examination of the pus and the discovery of the ray-fungus is the diagnostic feature. When the lesion is in the region of the throat or of the udder examination of the lymphatic glands of the part may afford considerable assistance in that they are rarely affected in actinomycosis but are constantly affected in tuberculosis. The presence of discharging sinuses with a gritty pus points to actinomycosis, but may also occur when tuberculous lesions have undergone some degree of calcification. This, however, is uncommon since calcified lesions rarely break down and discharge pus. The use of tuberculin may assist in the case of a negative reaction excluding tuberculosis, but a positive reaction, while showing that the animal is affected with tuberculosis, would not necessarily indicate that the particular growth was tuberculous. Actinomycosis may also be distinguished by the improvement usually manifested when the patient is submitted to the iodide treatment. *Actinobacillosis*, which is somewhat rare, resembles actinomycosis in many clinical features. It more often, however, involves the lymphatic glands of the part while the jaw-bones are rarely affected. On microscopic examination of the pus colonies of clubs may be demonstrated in each case. The organism stains by Gram's method in actinomycosis but not in actinobacillosis.

In the horse, actinomycosis may be confused with glanders, a streptococcus infection of the tongue, or botriomycosis. It is only likely to be confused with *glanders* in the early cases, for in that affection the submaxillary enlargements never attain such large dimensions and rarely burst. The demonstration of the ray-fungus on the one hand or a positive reaction to mallein on the other hand affords positive diagnosis. In *streptococcic infections of the tongue* that organ rapidly becomes enlarged and protrudes from the mouth, an acute glossitis being set up. Small abscesses may be set up in the pus of which numerous streptococci can be demonstrated. This condition has been erroneously diagnosed as actinomycosis, but that disease is practically unknown in the tongue of the horse, and in whatever situation it occurs it always assumes a chronic course. *Botriomycosis* must be distinguished by microscopic examination of the pus from the lesion whether it be involving the spermatic cord or elsewhere. Moreover, this condition is quite common in the horse, while actinomycosis is comparatively rare.

Treatment.—Surgical treatment is sometimes called for, particularly in the case of pedunculated growths in the pharynx, which must be removed by means of an ecraseur. Circumscribed actinomycotic growths of the skin or in subcutaneous situations may also be excised. Large inoperable growths may be incised deeply and curetted, and injected with tincture of iodine. An affected tongue should not be interfered with surgically.

The best results are obtained by medical treatment, potassium iodide being a specific for the disease in cattle and almost as effective in the horse. The dose should be regulated according to the size of the patient, and should be about one-quarter of a drachm for each hundred pounds body weight, that is, from two to three drachms once daily. It is best given dissolved in about a pint of water or linseed gruel and drenched, in order to ensure the animal getting the whole of it. After about a week the animal may show signs of iodism in the form of diminished appetite, catarrh of the nasal mucous membrane and conjunctiva, and the fæces dry and maybe coated with mucus. The potassium iodide must then be stopped for two or three days. During this interval 4 grains of biniodide of mercury dissolved with the assistance of 10 grains of potassium iodide may be given with advantage, as the growth otherwise increases somewhat when the iodide is stopped. The course of treatment varies apparently according to the susceptibility of the animal to iodism. Improvement is most marked in those animals showing signs of iodine poisoning and in them, with proper intervals, a period of four to six weeks will generally see recovery complete. In other cases a longer time may be necessary, while in some cases the patient appears to be immune to the effects of iodine, and treatment fails. In favourable cases the enlargement of the tongue or the tumour gradually subsides and disappears, leaving only a small cicatrix to mark its previous existence if there has been a granulating skin wound. These good results, however, are not so well shown when the bones are invaded, as the damage to these structures is not so easily repaired. It is doubtful if the case is worth treating if the swelling of the bone much exceeds the size of a goose egg. Biniodide of mercury is sometimes used throughout as a substitute of potassium iodide; the daily dose should be five to eight grains dissolved with the assistance of ten grains of potassium iodide, and administered as a drench in a pint of water. Although fairly good it is slow in action and its effects are not so generally satisfactory.

When there is ulceration or abscess formation these conditions must be treated accordingly, by curetting and syringing, or packing the cavities with wool saturated with iodised phenol or Lugol's solution.

It would be very interesting to know exactly how the iodides produce their effects. I have observed in some cases that more or less acute inflammatory phenomena, such as heat, redness, and increased pain, are set up in the lesions after a few days' administration of the drug. In addition to the effects on the diseased parts potassium iodide, when administered for a long period, produces emaciation and glandular atrophy. This latter point should be particularly borne in mind in the cases of males required for stud purposes, as it is said to produce sterility. These ill effects, however, can be largely avoided by regulating the doses and intervals and seeing that the patient gets plenty of nutritious and easily digested food. Where the tongue is affected to more than a slight degree, liquid food, as milk and thin gruel, must be given, and it may be necessary to administer them by bottle. Great care must be taken in drenching these animals, as choking followed by pneumonia may easily occur owing to the difficulty in swallowing. Another word is necessary with reference to milch cows. By the administration of potassium iodide the milk yield is considerably reduced and some of the iodine is excreted by the mammary gland. Consequently the milk is tainted and may be rendered temporarily unfit for food.

G. H. W.

ACTINOBACILLOSIS

Actinobacillosis is a disease of cattle occurring principally in South America but which is also met with in Europe and in North America. It is characterized by symptoms closely resembling those of actinomycosis, from which it cannot be positively distinguished clinically.

Etiology.—Actinobacillosis is caused by a small organism called the *actinobacillus* which in the tissues develops into small colonies which may attain the size of a small pin's head resembling the colonies of actinomycosis and consisting of small clubs. The bacilli do not stain by Gram's method and are thus distinguished from the filaments of actinomycosis.

Symptoms.—As already stated the symptoms closely resemble those of actinomycosis. They occur especially in the skin and the subcutaneous tissues in the region of the throat, in the tongue—setting up "wooden tongue"—and in the pharynx in the form of polypi. The udder is also occasionally affected. The lymphatic glands of the affected part are practically always involved and enlarged. Affected animals lose condition mainly owing to the mechanical interference with feeding.

Treatment.—This affection is usually amenable, though not so readily, to the same measures as are recommended for actinomycosis, particularly the internal administration of potassium iodide and the treatment of the local lesions or abscesses with iodine solutions

G. H. W.

MALIGNANT CATARRH OF THE OX

Gangrenous Coryza ; Malignant Catarrhal Fever of Cattle

Malignant Catarrh is a contagious affection of cattle characterized by acute catarrhal inflammation of the mucous membranes of the respiratory and alimentary tracts, and sometimes the skin. The affection is not rapidly contagious, and it commonly happens that cases are sporadic, although it may assume an enzoötic character. In some places it is a permanent enzoötic, and may break out in several successive years.

Etiology.—Although the affection is undoubtedly contagious the actual causal organism is yet unknown. Numerous bacteria may be met with in the lesions, but none of them have been shown to be specific for this affection. The natural method of infection is not definitely known ; but experimentally it has been produced by way of the alimentary tract. The affection is not uniform in its occurrence, and several forms are referred to, such as (*a*) the *nasal or respiratory* ; (*b*) the *abdominal*, and (*c*) the *cutaneous form.* The nasal type is the most common, but sometimes all three forms occur simultaneously.

Symptoms.—In general, malignant catarrh affects the mucous membranes of the head and the respiratory organs, and of the alimentary tract, producing diphtheritic lesions, and is associated with brain disturbance. The disease develops rapidly with rigors and high fever. There is very great depression, and the animal can scarcely support his head. There is a staring coat and the back is arched, while the base of the horns is hot to the touch. There is very rapid loss in condition. Conjunctivitis is set up with closure of the eyes, opacity of the cornea, and a discharge at first lachrymal and later catarrhal. In bad cases the whole eye may become involved, producing ophthalmia and permanent blindness. Catarrh of the nose is observed about the same time. The mucous membrane is deep red or purplish in colour, and the discharge is usually streaked with blood—in the later stages may contain shreds of mucous membrane and become offensive in odour. The affection often extends into the horn core from the sinuses of the head, and may result in shedding of the horns. At the same time, inflammation may extend to the larynx and trachea, causing difficult respiration, roaring, and coughing, while spasms of partial suffocation may be set up and may even produce asphyxia. In other cases, the digestive apparatus becomes involved, the mouth is hot and dry, and may become ulcerated, setting up dribbling and a very offensive breath. Ulcers may be observed on the gums and dental pad, and inside the cheeks. Constipation occurs during the early part of the affection, but may be alternated with diarrhœa. In the latter case, the fæces are very offensive, may be blood-stained and contain shreds of mucous membrane. Sometimes the kidneys and bladder become involved, setting up nephritis and cystitis, and abortion may be brought about in pregnant cattle. When the skin is affected in the exanthematic form vesicles and ulcers may be produced, and sometimes large sloughs. Occasionally the hoofs are shed.

Brain symptoms are frequently exhibited, and usually consist of excitement in the earlier stages, followed by coma and collapse. Death is usually preceded by subnormal temperature.

Prognosis is not very hopeful, the average mortality probably exceeding 50 per cent. In severe cases death may occur in from three to five days. When recovery occurs the course of the disease is usually from three to four weeks. In some cases it may be extended to twice that period. In any case of recovery the period of convalescence is protracted.

Differential Diagnosis.—The affection most likely to be confused with malignant catarrh is rinderpest. The main points of distinction are the markedly contagious nature of rinderpest and its very rapid course. Moreover, in rinderpest the abdominal symptoms are predominant, while in malignant catarrh the respiratory form is the more common.

Treatment.—Although the affection is not rapidly contagious, isolation of affected animals and every precaution against contagion should be adopted, and thorough disinfection of everything that might become contaminated with discharges from affected animals must be carried out.

Curative measures are not very satisfactory. All exposed lesions should be disinfected with strong disinfectants such as carbolic acid 5 per cent or chloride of zinc grs. x. to ℥i., and the nasal chambers should be irrigated with weaker solution. In former times the tips of the horns were sawn off to facilitate the irrigation of the sinuses and the nose. The eyes should be frequently douched with boric acid gr. x. to ℥i. or other mild antiseptic lotions, to which a little opium may be added. Fumigations with eucalyptus or terebene are also useful.

Internally, a combination of quinine and iron is indicated, while salicylates and potassium iodide may also be administered. General stimulants may also be necessary, while in cases of diarrhœa intestinal astringents such as opium or catechu should be given. The appetite should be tempted with a laxative, nourishing diet, which should be concentrated, and easily digestible, such as linseed mashes, oatmeal gruel, and milk.

G. H. W.

HÆMORRHAGIC SEPTICÆMIA
(PASTEURELLOSIS)

Under the term "hæmorrhagic septicæmia" are grouped a number of diseases, occurring in almost all the domesticated animals, rodents, fowls, and certain wild animals, which are characterized by hæmorrhages into the tissues and the presence of a particular type of organism in the blood-stream and tissues of the body.

Pasteur (1880) first described a small ovoid bacillus staining deeply at its poles as the causal agent of fowl cholera, hence similar organisms from other sources are frequently referred to as being of *the fowl cholera type.* Later, rabbit septicæmia (Gaffky, 1881), Bollinger's Wildseuche (Kitt, 1883), swine plague (Lœffler, 1885), and fowl cholera were placed by Hueppe (1886) into one group which he called the "hæmorrhagic septicæmias."

Other diseases ascribed to varieties of the same organism are : pleuro-pneumonia of calves (Poels, 1886), barbone of buffaloes (Oriste and Armanni, 1886), pneumo-enteritis of sheep (Galtier, 1889), pleuro-pneumonia (? influenza) of horses (Lignières, 1900), canine distemper and typhus (Lignières), white scour and pneumonia of calves (Nocard), pneumonia of goats (Nicolle and Refik Bey, 1896), and entéqué (Lignières, 1898).

Nocard and Leclainche first applied the term "*bactéridie ovoïde*" to the bacilli of the group, but later they and French bacteriologists generally adopted the classification of Lignières, who supported Trevisan's suggestion to name the genus "*pasteurella*" and distinguished the varieties as *Pasteurella bovis*, *equi*, *ovis*, *gallinæ*, *canis*, etc., the diseases being known as "*pasteurelloses.*" Kitt proposed the name *Bacillus plurisepticus* for the organisms of this group, and Hutyra and Marek employ the term *Bacillus bipolaris septicus*, designating the different varieties according to the species of animal affected as *Bacillus avisepticus*, *Bacillus bovisepticus*, etc.

It is now known that some at least of the organisms included in this group are only secondary invaders of the tissues of affected animals and not the primary cause of the diseases attributed to them ; thus, a bipolar organism as the causal agent of distemper is now discredited, while *Bacillus suisepticus* is regarded as a normal inhabitant of the healthy pig which only produces pathogenic effects when invasion of the body by the virus of swine-fever occurs. Non-pathogenic species of pasteurella have been isolated from various sources, and it is certain that some of the pathogenic varieties can live in soil and water, starting outbreaks of disease on gaining entrance to the bodies of susceptible animals.

The differentiation of the various members of the group is far from satisfactory, but in the case of the more important diseases classed as hæmorrhagic septicæmias or pasteurelloses the etiological rôle of the bipolar organism is firmly established, and from the close similarity between the organisms from various animals it is natural to regard them as varieties of the same bacillus. The pathogenicity of the different strains varies, however, according to their origin, but the virulence of any strain can be altered by repeated re-inoculations into animals or birds, highly susceptible species increasing, and more resistant species reducing, the virulence.

In their general characteristics the organisms of the hæmorrhagic septicæmia group exhibit great similarity; the variations comprise slight differences in size and rapidity of growth on artificial media and of pathogenicity for laboratory animals, but are not sufficiently well marked to render separate descriptions necessary.

Morphology and Biology of the Organism.—As found in the body tissues, the organism of hæmorrhagic septicæmia appears as a short bacillus with rounded ends from 1 to 2 μ long and about 0·5 μ broad, though it varies slightly in size according to its origin.

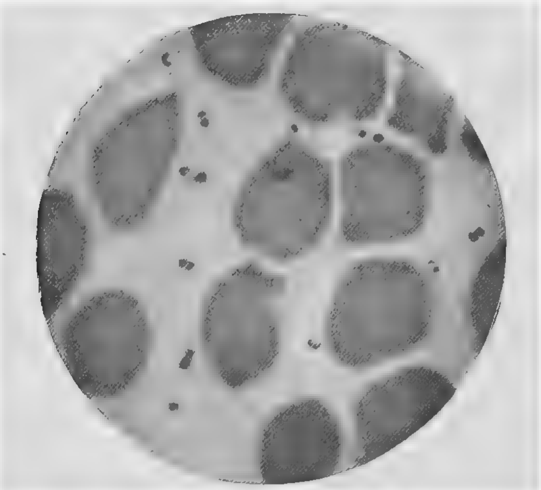

FIG. 19.—Hæmorrhagic septicæmia of buffalo; *Bacillus bubalisepticus.* Smear from blood of rabbit; stained dilute carbol fuchsin and washed with 0·5 per cent acetic acid. (Magnified 2000 times.)

In young cultures rounded forms resembling cocci are numerous, while in older and less virulent cultures larger bacillary forms, sometimes short threads, are seen. The bacillus is non-motile and does not form spores. Any of the aqueous aniline dyes stain the organism readily when, unless deeply stained, it shows a clear unstained area in the centre, the so-called bipolar staining; this appearance is best seen in preparations from the tissue fluids. Leishman and Giemsa stains give good results, also methylene blue or dilute carbol fuchsin, the latter after slightly decolorizing with ½ per cent acetic acid. The organism is not stained by Gram's method.

A pure culture of the organism can usually be obtained from the blood in acute septicæmic cases. The bacillus grows readily on most culture media, except potato, in the presence of oxygen, at temperatures between 28° and 37° C., but prefers media with slightly alkaline reaction and those containing blood serum. On the surface of gelatine or glycerine agar the colonies appear within twenty-four hours as small transparent drops which later become white, opaque, and viscid; gelatine is not liquefied. Peptone or serum broth becomes turbid, but later the growth settles as a slimy sediment. Alkaline media become acid; no gas is formed, but old broth cultures have a peculiar sour odour; milk is not coagulated. Surface growths should be subcultured every five or six days, before the water of condensation has dried in the tube, otherwise vitality may be lost.

Filtrates of broth cultures of the bacillus from fowls and pigs are said to be highly toxic, but the formation of toxins in culture media is not a constant character of the hæmorrhagic septicæmia organisms.

The bipolar bacilli possess feeble powers of resistance outside the body, being easily destroyed by heat, desiccation, sunlight, and dilute solutions of antiseptics.

In moist soil and water, however, these organisms are able to live for long periods, and though they lose a good deal of their pathogenicity by this saprophytic existence yet on gaining entrance to susceptible individuals, under certain conditions, their virulence may become restored; in this way sporadic outbreaks of the disease probably arise.

The rabbit and mouse are highly susceptible and die in from twelve to thirty-six hours after the injection of a small amount of culture; guinea-pigs are more resistant to some strains.

The larger domestic animals show great variation in their susceptibility; organisms from some species of animal being quite specific in their pathogenicity.

Intravenous inoculation of culture produces the most rapid results, while subcutaneous injection gives rise to an extensive œdematous swelling followed very shortly by systemic effects in susceptible animals.

The feeding of virulent material by the mouth, except in the case of fowl cholera, only exceptionally gives rise to the disease; the digestive tract is, however, regarded as the probable channel of entrance of the organism in natural outbreaks, since it has been shown that infection may occur in this way when the resistance of the animal has been reduced by various means. Natural infection may also occur through abrasions of the skin or by inhalation in some cases. The organism may be conveyed by biting flies (hæmorrhagic septicæmia of cattle and wild game) or lice (fowl cholera) in the same way that *Bacillus pestis*, the cause of bubonic plague and an organism of the same type, is transmitted from infected rats to man by the rat flea.

The pathological changes caused by the bipolar bacilli vary greatly in different animals, but they follow a very similar course in each type of the disease. The very acute cases are characterized by high fever, depression, sometimes diarrhœa, and death about twenty-four hours after the onset of illness; in the less acute forms the disease may last a few days, when the mucous and serous membranes become inflamed, hæmorrhagic exudates collect in the body cavities and subcutaneously, and in some cases pneumonia is the most prominent feature. Whenever the disease assumes a chronic course, the lungs are the organs particularly affected. A constant post-mortem lesion in all but chronic cases is the presence of small hæmorrhages into the tissues and under the mucous and serous membranes.

In all acute cases the organism is found in great numbers in the blood and body fluids.

Following on the discovery by Pasteur that attenuated cultures of the fowl cholera bacillus produce immunity against that disease, vaccines have been prepared for most of the diseases of this group. Kitt, Jensen, and Lignières have shown that vaccines of one strain will also give protection against other diseases of the group, though to a lesser extent than in the case of the particular variety from which they have been prepared. Lignières prepared a polyvalent vaccine from mixed cultures of bacteria from cattle, horses, sheep, dogs, pigs, and fowls with which it is claimed all these species of animals can be given a serviceable immunity lasting about a year.

By injecting animals with repeated doses of the fowl cholera bacillus Kitt (1897) obtained serum from them which protected fowls against this organism, and subsequently sera for several diseases due to bipolar bacilli were similarly prepared. As in the case of the vaccines the sera conferred a certain immunity against other varieties of the group, and Lignières and Spitz, by the injection of horses with cultures of the same six bipolar bacilli used in the vaccine, obtained a polyvalent serum against all the hæmorrhagic septicæmias.

Hæmorrhagic Septicæmia of Bovines and Buffaloes

Synonyms.—Malignant sore throat, infectious broncho-pneumonia. Pasteurellosis bovis (*French*); Wild- und Rinderseuche (*German*); Barbone bufalino (*Italian*); Ghotwa (*Hindustani*).

History.—The disease was first described by Bollinger in 1878 as occurring in wild deer at Munich and later in cattle, horses, and pigs; the causal organism was discovered by Kitt in 1885. The disease in buffaloes (" Barbone ") was described by Oreste and Armanni (1886). More recently numerous investigators have studied the disease in the Argentine, United States, Philippine Islands, and India, as well as in Europe. The existence of the disease in the British Isles has not been established.

Occurrence.—The disease is most prevalent in low-lying lands subject to periodical inundation, but may occur sporadically under any conditions. In India the most serious outbreaks are experienced during the monsoon rains, but rain at any season of the year usually gives rise to cases in affected districts.

Etiology.—The cause of the disease is the bipolar organism, *Bacillus bovisepticus* (Kruse) or *Pasteurella bovis*; when found in buffaloes, *Bacillus bubalisepticus* (Hutyra and Marek). The characters of the organism are those typical of the group.

Pathogenicity.—Buffaloes are very susceptible and outbreaks of the disease are most common in these animals; cattle may become affected at the same time or independently. The virulence of the contagion in the bovine form of the disease varies considerably, but cultures of the organism are less pathogenic for cattle than the buffalo strain. In America 1 c.c. of unattenuated broth culture of *Bacillus bovisepticus* is employed for the vaccination of cattle, much larger doses being necessary to cause a fatal attack of the disease, but 0·1 c.c. of broth culture of the buffalo strain (India) kills both cattle and buffaloes in from twenty-four to forty-eight hours by subcutaneous injection.

Pigs, horses, and elephants are occasionally attacked, and wild herbivora may develop the disease naturally. Rabbits are very susceptible to artificial infections, also, to a less extent, mice, guinea-pigs, rats, and pigeons. Camels, sheep, and goats require the injection of a considerable dose of culture to cause death. Dogs, fowls, and ducks are resistant.

Young animals are more susceptible than old to both natural and artificial infections. After subcutaneous inoculation of infective material an œdematous swelling quickly forms at the point of injection, followed in a short time by symptoms of general intoxication and death. The organism can be recovered from every tissue and fluid of the body and is excreted in the fæces, urine, saliva, and milk.

Natural infection may occur through wounds of the skin or mucous membrane of the mouth. The fact that the administration by the mouth of large doses of virulent culture of the organism rarely produces the disease, except in rabbits and guinea-pigs, has been taken as proving that ingestion cannot be a common mode of natural infection, but experiments have shown that when the resistance of the animals is reduced by exposure, insufficient food, fatigue or other cause, infection through the uninjured mucous membrane of the digestive tract may occur. The early appearance of œdematous swellings round the throat in most cases is regarded as indicating infection through the pharyngeal membrane.

Sporadic cases and the first animals attacked in an enzoötic outbreak probably acquire the infection from soil or water, as a result of lessened resistance from some cause; subsequently the disease may be transmitted by biting flies (*Simulium ornata* was incriminated by Dammann and Oppermann), ticks or lice, or by ingestion or inhalation of the infective discharges of the sick animals. The disease, however, is not actively contagious and nothing is known as to the existence of "carriers."

Symptoms.—After the inoculation of virulent material the temperature rises and the local swelling develops in from six to eighteen hours; under natural conditions the period of incubation is probably from one to two days.

The earliest symptom is a rapid rise in body temperature to 41° or 42° C. (106° to 108° F.), quickly followed by cessation of feeding and rumination and general depression; the pulse and respiration become accelerated, visible mucous membranes injected, and saliva flows from the mouth. At first there is constipation, but this is soon followed by diarrhœa, with signs of colic and straining; later the fluid fæces may contain flakes of mucus or blood.

The subsequent course of the disease varies in different breeds of animals, outbreaks, and countries, and several forms have been described; thus cases may be classed as *acute*, *subacute* and *chronic*, or according to the localization of the principal lesions as *cutaneous* or *œdematous*, *pectoral*, and *intestinal*, but intermediate and mixed cases are frequently met with. In many of the acute cases death occurs before any well-marked symptoms are observed.

The Œdematous Form.—This is the commonest type, especially in buffaloes. Closely following the earliest symptoms, a hot painful swelling appears in the region of the throat, spreading to the intermaxillary space, cheeks, neck, or dewlap and sometimes to the forelegs and shoulders, at first doughy but later very tense. The head is extended and a short painful cough may occur during the early stages, followed by stertorous breathing and difficult deglutition. The tongue becomes swollen and protruding, its mucous membrane purple or reddish brown in colour, and tenacious saliva flows from the mouth; there is conjunctivitis with escape of tears down the face and yellowish mucous discharge from the nostrils. Death may occur as the result of suffocation from six hours to two days after the onset of symptoms, preceded either by stupor or a short period of restlessness. In the more prolonged cases the digestive tract or lungs become involved, with signs of colic and diarrhœa or laboured breathing and frequent cough. Cattle affected simultaneously with buffaloes usually exhibit similar symptoms. Pigs appear to be especially liable to contract the disease from buffaloes, and in them also swelling of the throat region and pharyngitis are usually pronounced.

The Pectoral Form.—In these cases the hurried breathing and suffocative cough arise from the implication of the lungs and pleura. The animals stand with legs fixed, head extended, nostrils dilated, and mouth open; a colourless or reddish frothy discharge flows from the nose. Examination of the chest reveals consolidation of lung tissue or fluid in the pleural cavities. Young animals usually die rapidly, but in other cases death may not occur for four or six days; in cases which recover the lungs are usually permanently affected. The disease as it is met with in the American stock-yards and on board ship is commonly of the pulmonary type.

The Intestinal Form.—This is usually associated with the œdematous or pectoral types which survive longer than a day. The symptoms are restlessness, colic, diarrhœa or dysentery, with straining and inflammation of the mucous membranes.

A chronic type of hæmorrhagic septicæmia, **Entéqué,** of cattle is described by Lignières as occurring in the Argentine; horses and sheep are also occasionally attacked. Two forms are described, an intestinal form, in which there is persistent diarrhœa and wasting, the animal dying in from three weeks to four months, and a wasting form, characterized by emaciation, anæmia, and frequently inflammation of the joints; these cases may live for some years. In the latter form calcifications in the lungs and walls of the large arteries are the outstanding lesions in cattle, but these changes are not observed in horses or sheep. The causal organism is said to be a small form of bipolar bacillus.

Course and Prognosis.—The course varies from a few hours in the most acute cases to several days when the lungs are primarily affected. In the chronic forms the duration of the disease may extend to months or years.

From 50 to 95 per cent of the animals attacked succumb; mortality is higher in buffaloes and carabaoes than in cattle.

Lesions.—These vary considerably according to the type of the disease.

In the œdematous form there is an infiltration of the subcutaneous tissues of the head, throat, and neck with sero-gelatinous fluid, either clear and yellowish in colour or blood-stained. Similar infiltrations may be found under the tongue and surrounding the pharynx and larynx. The tongue is enlarged, infiltrated with yellow fluid, and studded with small hæmorrhages.

The mucous membrane of the fourth stomach and small intestine shows acute hæmorrhagic inflammation; the large intestine is less markedly affected. The contents of the canal are fluid, yellowish-grey in colour or mixed with blood. The lesions of the digestive tract are especially marked in the so-called intestinal form of the disease.

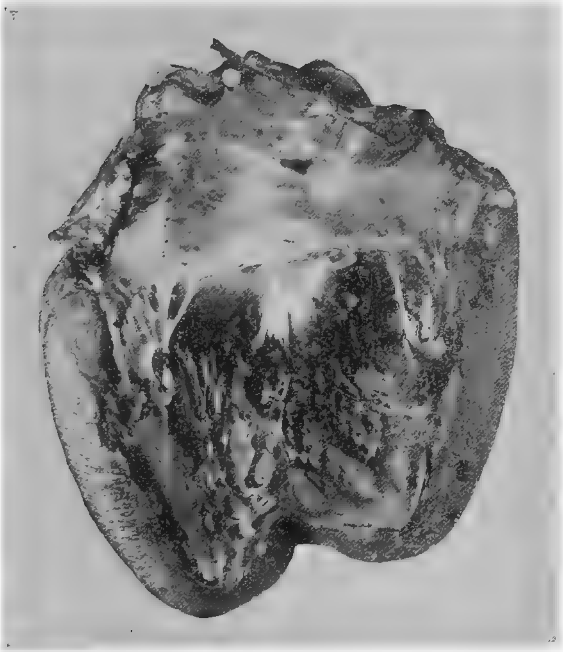

FIG. 20.—Hæmorrhagic septicæmia. Heart of buffalo showing hæmorrhages under the endocardium.

In the pectoral form one or both lungs contain areas of hepatization, hæmorrhagic or grey, while the septa between the lobules are thickened and infiltrated with yellow serous fluid which escapes on incising the organ; the appearance of the cut surface of the lung may closely resemble that seen in contagious pleuro-pneumonia except for the similarity of the age of the affected lobules.

In any of the forms assumed by the disease the pleural and peritoneal cavities may contain an excess of fluid, either yellow or reddish in colour, while the serous membranes are usually studded with numerous small hæmorrhages. The pericardial sac is distended with fluid, and minute hæmorrhages may be present on the surface and throughout the muscular tissue of the heart, but the most constant and almost diagnostic post-mortem lesion of the disease is the presence of blood extravasations under the endocardium, especially of the left ventricle. The lymphatic glands of the body are enlarged, œdematous and sometimes hæmorrhagic, but the spleen is usually normal.

In the chronic cases the lungs show an excess of fibrous tissue and frequently areas of necrosis or caseation.

The presence of gelatinous infiltrations into the connective tissues and small hæmorrhages under the serous membranes are the most characteristic lesions of the disease.

Differential Diagnosis.—Hæmorrhagic septicæmia has to be differentiated from the following diseases:

Anthrax.—Those cases which show swelling of the throat and signs of enteritis before death may closely simulate the œdematous form of hæmorrhagic septicæmia; microscopical examination of the blood and fluid from the swellings may reveal the anthrax bacillus, but the organism will be found with much greater certainty after death. The enlargement of the spleen and tar-like condition of the blood should raise a strong suspicion of anthrax. If the presence of large numbers of putrefactive organisms makes the microscopical diagnosis uncertain, rabbits or guinea-pigs may be inoculated.

Black Quarter.—One or other of the limbs are usually swollen in black quarter and the skin over the swelling is cold and crepitates on pressure. On cutting into the swelling the muscles are affected, being blackened and full of bubbles of gas. Microscopically the sporulating bacillus will be found; the inoculation of rabbits and pigeons with black quarter tissue usually fails to infect.

Malignant Œdema.—The swelling in this disease may crepitate and usually results from deep wound infection; cases are generally single. The long bacillus is present in the œdema; intravenous inoculation fails to infect test animals.

Cattle-plague.—Œdematous swellings are absent in cattle-plague, while mouth lesions are frequently present. The history of infection, rapid spread, post-mortem lesions, and failure to infect small laboratory animals with material from suspected cases enable this disease to be distinguished from hæmorrhagic septicæmia.

Contagious Pleuro-pneumonia.—The lung lesions in this disease are very similar to those found in the pectoral form of hæmorrhagic septicæmia, but differ in that the affected lobules are in various stages of the pathological changes, as shown by differences of colour, whereas in hæmorrhagic septicæmia larger portions of the lungs are involved and all to the same degree. The course of pleuro-pneumonia is slow, only bovines are susceptible, the lesions are confined to the lungs, and the bipolar bacillus is absent from the tissues.

Cornstalk Disease.—This disease occurs in America among cattle which are allowed to graze on the cornstalks during late autumn; it is characterized by a very rapid course and hæmorrhages into the serous membranes. Billings (1889) found bipolar bacilli in the lung lesions and identified the disease with the hæmorrhagic septicæmias; Nocard (1890) also isolated a similar organism from the lungs of American cattle landed in France. Later investigations by Moore (1892), however, showed that the disease was not microbial but due to the presence in the stalks of a toxic substance, which Mayo states to be potassium nitrate in excess. If the stalks are cut early, the injurious effects are said to be prevented. It is probable that the two diseases are frequently confused, but on microscopical examination of the blood and body fluids the bipolar bacillus will be found in cases of hæmorrhagic septicæmia and not in cornstalk disease.

Protective Inoculation. (1) *By means of Vaccines.*—Oreste and Armanni (1887) attenuated the *B. bubalisepticus* by passage through pigeons and employed the blood of these birds, after they had died from the inoculation of the organism, for the immunization of buffaloes; three injections of 0·1 c.c. of the blood were given.

By growing the organism at a temperature of 30° to 32° C., Oreste and Morcone (1889) reduced its virulence so that cultures could be employed to give protection to buffaloes and sheep.

Lignières (1902) prepared a polyvalent vaccine with strains of the hæmorrhagic septicæmia bacillus from cattle, sheep, horses, pigs, fowls, and dogs. Cultures of the organisms of uniform virulence are kept at a temperature of 42° to 43° C. for five days to produce the first vaccine and for two days for the second vaccine.

The vaccines are injected subcutaneously in doses of from 0·15 to 1 c.c. according to the size of the animal, at an interval of twelve to fifteen days and are said to give protection against the disease in all species of animals for a period of about one year.

Mohler and Eichhorn (1912) employed vaccines prepared by Lignières' method for protecting the buffalo herd in the Yellowstone Park against hæmorrhagic septicæmia, which was causing a heavy mortality. The animals were injected subcutaneously with 1 c.c. of each vaccine at an interval of ten days, and no further cases of the disease occurred during the succeeding twelve months.

These investigators also tested the immunizing power of vaccines prepared in the same way as those of Lignières but to which ·5 per cent carbolic acid had been added, thus making them dead vaccines. They found that the carbolized vaccines gave protection against an inoculation of the virulent organism, but, as judged by complement fixation tests of the serum of the treated animals, this protection was of shorter duration than after injections of the uncarbolized vaccines. During recent years similar vaccines have been frequently employed in America with satisfactory results.

Holmes tested the immunity conferred by dead vaccines prepared in various ways from cultures of the organism. Broth culture of a virulent strain of the organism heated to 65° C. for half an hour and carbolized was found to protect young cattle and buffaloes, when injected subcutaneously in doses of 5 to 10 c.c., for a period of six weeks against an inoculation of the virulent organism. The vaccine is harmless, only causing in some cases a slight swelling which disappears in a few days; protection is not established until four days after the injection of the vaccine. About 150,000 doses of vaccine are employed annually in India as a prophylactic measure in areas where the disease is enzoötic and periodic, inoculations being carried out at the beginning of the winter rains and monsoon season.

(2) *By means of Immune Serum.*—Following the observations of Kitt (1897) and others regarding immune serum against fowl cholera, Lignières and Spitz (1902) showed that the same principle applied to all diseases of the hæmorrhagic septicæmia group, and they prepared a polyvalent serum from horses by the injection of strains of the organism from six species of animal. This serum is used both as a prophylactic and for the treatment of the disease.

Blin and Carrougeau (1902) produced an immune serum against the disease in cattle and buffaloes by the immunization of horses. Lingard (1903) hyper-immunized bovines and buffaloes themselves and found their serum protective for susceptible animals in doses of 5 to 10 c.c. against an injection of virulent culture. Later, Holmes (1909) improved the procedure for preparing the serum, and about 3000 litres are now employed annually in India.

The method followed at the Muktesar laboratory is briefly as follows: Buffaloes and hill cattle are first injected subcutaneously with a protective dose of immune serum and four hours later receive 0·1 c.c. of forty-eight-hours-old broth culture of virulent hæmorrhagic septicæmia bacilli; the initial protection may also be conferred by an injection of dead vaccine followed in ten days by 0·1 c.c. of virulent culture. A temperature reaction and local œdema usually follow the inoculation of the culture; when this has subsided, after eight to ten days, an injection of 1 c.c. broth culture is given. Further injections of 5 c.c., 25 c.c., 100 c.c., and 500 c.c. of culture are given at intervals of ten days, and the animals are then bled for serum ; the injections and bleedings are repeated, the dose of culture being gradually increased up to 1000 c.c.

An injection of the serum confers an immediate immunity lasting for a period of three or four weeks. It has been found very effective when employed during outbreaks of hæmorrhagic septicæmia, the mortality being at once arrested ; the serum also possesses some curative power.

(3) *By means of Serum and Culture.*—Blin and Carrougeau (1902) immunized cattle and buffaloes by giving injections of immune serum and 0·1 to 0·5 c.c. of virulent culture simultaneously, but claimed better results from the subcutaneous injection of a mixture of 20 c.c. of serum and 2 c.c. of culture.

De Giaxa and Di Donna (1910) report the immunization of buffalo calves by two simultaneous inoculations of culture (1 c.c.) and serum (15 c.c.) at an interval of ten days.

The method found most satisfactory by Holmes (1908) was that which is now followed when immunizing animals intended for serum making, namely, to give the injection of culture (0·1 c.c.) four hours after the dose of serum ; but owing to the risk involved it is not applicable in the field in India.

(4) *By means of Culture or Toxin.*—Hardenbergh and Boerner (1916) found that 1 c.c. of forty-eight-hours-old culture of *B. bovisepticus* failed to produce the disease when injected into calves, and employed this dose as a vaccine against the disease.

In India as small a dose as 0·02 c.c. of forty-eight-hours-old broth culture of the hæmorrhagic septicæmia bacillus from either cattle or buffaloes may prove fatal on injection into these animals, and 0·1 c.c. is an almost certain lethal dose. Owing to this high susceptibility and the prejudice of the people against any inoculation attended with risk to the life of cattle, even attenuated cultures cannot be employed in that country.

Virulent broth culture of the organism when administered by the mouth even in large doses usually fails to cause the disease, but produces an immunity sufficiently strong to resist the inoculation of a lethal dose of culture in the majority of cases. Animals whose vitality has been reduced by fatigue, exposure, or other cause may, however, become infected when given culture by the mouth.

The filtrate from broth culture (toxin) when inoculated into susceptible animals confers protection and is harmless by subcutaneous injection ; the immunity is of short duration.

For the production of active and lasting immunity attenuated vaccines or the serum and culture method are most suitable, though neither is free from risk. When the disease appears only at certain seasons of the year, immunity of sufficient duration to protect the animals through the infective period may be produced without risk by the injection of dead vaccines. Serum, by giving immediate protection and arresting the mortality, is most suitable for use during an outbreak, a more lasting immunity being established later, if necessary, by the use of vaccines or serum and culture.

Treatment.—There is usually little opportunity of treating the more acute cases, and even in those running a slower course medicinal agents appear to exert but slight curative effect.

Holmes tested a large number of drugs, including carbolic acid, potassium permanganate, and other antiseptics, administered by the mouth, subcutaneously and intravenously. Iodine was the only agent which had a decidedly beneficial effect ; in doses of ½ to 1 drachm (2 to 4 grammes) with an equal quantity of potassium iodide, in 300 c.c. of water subcutaneously, or double these doses by the mouth, a large proportion of recoveries was obtained in both cattle and buffaloes.

The injection of disinfectants, carbolic acid (3 per cent), or turpentine into the throat œdema is also recommended.

Immune serum in large doses (100 c.c.) intravenously is of value if given early in the attack.

Sanitary Measures.—Hæmorrhagic septicæmia of cattle and buffaloes is not usually an actively infectious disease in the sense that cattle-plague and foot-and-mouth disease are infectious, the contagion being usually obtained from the soil, so that strict quarantine regulations or the slaughter of affected animals do not appear to be necessary.

In America, however, the disease is sometimes carried to clean farms by animals which have become infected in the stock-yards, and in some States the application of quarantine measures is contemplated.

The affected animals should be isolated and all in-contacts removed to clean dry pastures or premises where they may be immunized by one of the methods described. Infected buildings should be thoroughly disinfected, all litter and the carcases of any dead animals burnt.

Pigs should also be removed from contaminated areas since they may become infected from cattle or buffaloes.

Hides that have been completely dried are incapable of spreading the disease.

Hæmorrhagic Septicæmia of Sheep

Catarrhal Fever ; Pneumo-enteritis of Sheep ; Lombriz ; Pasteurellosis ovis

Several outbreaks of disease among sheep have been described in various countries, differing slightly in their characteristics but all having an organism of the hæmorrhagic septicæmia group as their essential causal agent, the *Bacillus ovisepticus*.

The relationship of a bipolar staining organism to the disease in sheep was first established by Galtier (1889) in the Basses Alpes; these outbreaks were associated with swine-plague, possibly in conjunction with swine-fever. Later, several outbreaks of pneumo-enteritis in sheep were investigated in France and a similar organism isolated in every case.

Lignières (1898) found that the determining agent of the sheep disease "Lombriz" in the Argentine is an organism of the hæmorrhagic septicæmia group, although animal parasites of the lungs, liver, and alimentary canal act as predisposing causes by lowering the vitality of their hosts and aiding the entrance of the organism into the tissues.

More recently hæmorrhagic septicæmia has been observed in many countries and frequently causes heavy mortality. Lambs are especially susceptible and in them the disease is very acute; older animals when attacked suffer from a more chronic form.

The causal organism possesses the characteristics common to the hæmorrhagic septicæmia group; it is easily isolated from the blood and organs in acute cases of the disease, but may be difficult to find in the chronic forms.

Natural infection probably results from ingestion of contaminated food or water. Influences which reduce vitality, such as weaning, exposure to damp or cold, and internal parasites, increase susceptibility; sheep on low-lying marshy ground are most frequently attacked.

Symptoms.—In the acute form the symptoms are high fever, loss of appetite, nasal discharge, rapid respiration, signs of abdominal pain, and death in from a few hours to two or three days. Less acute cases may live for two or three weeks; there is a purulent discharge from the eyes and nose, cough and symptoms of broncho-pneumonia or pleuro-pneumonia and fœtid diarrhœa. The mucous membranes are congested with petechial spots, and patches of dark red discoloration may appear on the exposed areas of skin. The lips, cheeks, and tongue may swell and the mucous membrane become ulcerated.

The chronic form, seen usually in older sheep, is characterized by persistent cough with nasal discharge, chronic pleuro-pneumonia, swelling of the carpal and tarsal joints, diarrhœa, and gradual emaciation; abortion frequently occurs. The affection may last for several months and many cases recover.

The *lesions* in the acute and subacute cases are similar to those seen in cattle; broncho-pneumonia is almost invariably present and small blood extravasations under the serous membranes; the pleural fluid may be in excess, clear yellow or cloudy.

In the chronic cases the lungs show areas of hepatization, with necrotic foci and connective tissue formation, nodules varying in size from that of a pea to a hazel-nut may be scattered through the lung tissue; these have a connective-tissue capsule and contain dry or creamy yellowish pus; the pleuræ are thickened and purulent exudate may be present in the pleural cavities. In some cases anæmia and emaciation may be the only post-mortem manifestations.

The spleen is usually normal in appearance, thus distinguishing cases from anthrax or piroplasmosis.

Protective inoculation has been practised with good results. Lignières employed both his polyvalent vaccine and polyvalent serum successfully.

Miessner and Schern prepared an immune serum from horses by injecting them with cultures of the *Bacillus ovisepticus*; this was both protective and curative. But a simultaneous injection of the serum and a bacterial extract was found most satisfactory; this conferred immunity for six weeks, which period could be extended by further inoculations of the extract.

Medicinal treatment has not proved successful. Chronically affected animals should be killed; the remainder of the flock, after removal to clean dry pastures, should be immunized with serum or serum and vaccine.

Hæmorrhagic Septicæmia of Pigs[1]

Swine-Plague; Schweineseuche (*German*)

The organism of the hæmorrhagic septicæmia group found in pigs, the *bacillus suisepticus* or *pasteurella suis*, has long been credited in Germany and America with the causation of a specific contagious disease.

In Great Britain this organism is regarded as a secondary infection in pigs suffering from swine-fever and responsible only for the lung lesions frequently encountered in outbreaks of that disease. This view is gaining acceptance also in other countries.

It seems reasonable to suppose, however, that the bipolar organism may exert its pathogenic effects in animals whose vitality has been reduced by causes other than the virus of swine-fever; and from the numerous outbreaks, usually sporadic, which have been reported unassociated with swine-fever, it would appear that hæmorrhagic septicæmia or swine-plague may occur in pigs as an independent disease.

Several observations have been recorded of pigs becoming infected during outbreaks of hæmorrhagic septicæmia in cattle and buffaloes, and conversely other species of animals may contract this disease from affected pigs.

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[1] See also p. 143.

The *Bacillus suisepticus* is easily isolated from the blood and tissues of affected animals and is similar to the other organisms of the group.

Symptoms.—The course of the disease varies. In the very acute cases there is high fever, loss of appetite, staggering gait, difficult respiration, hæmorrhagic spots over the body, hæmorrhage from the nose, anus, and urinary organs, and death in from twelve to twenty-four hours. Sometimes there is acute œdematous swelling of the throat. Other cases may live for from one to six weeks, with all the symptoms of acute pneumonia, short frequent cough, laboured breathing, pain over the chest, and nasal discharge. Red discolorations appear on the skin. Diarrhœa and sometimes dysentery set in before the end. The pneumonia may become chronic, and a few animals may recover although lesions remain in the lungs.

The *lesions* in the acute cases are those typical of hæmorrhagic septicæmia, small hæmorrhages throughout the body. In all cases which have been ill longer than twenty-four hours, lesions are constantly present in the lungs; these take the form of a multiple necrotic pneumonia. Areas of the lung tissue (lobules) are hepatized, dark red or greyish in colour, and studded with yellowish spots of necrosis. The interlobular septa are thickened by serous infiltration and the pleura over the affected areas is covered by fibrinous deposit. In chronic cases the necrotic foci may be surrounded by connective-tissue capsules. The mucous membrane of the stomach and intestines may show hæmorrhagic spots and catarrhal inflammation.

Owing to the frequency with which lesions due to the bipolar bacillus are associated with swine-fever, it is necessary to exclude this disease before a diagnosis of hæmorrhagic septicæmia can be established. In the absence of swine-fever, the disease is sporadic or occurs during an outbreak of hæmorrhagic septicæmia in cattle or buffaloes; it shows little tendency to spread rapidly through a herd as is the case with swine-fever. Preventive measures are similar to those necessary in dealing with the disease in other animals.

Many methods of protective inoculation against swine-plague by means of dead vaccines, toxins, attenuated cultures, and immune sera prepared from the *Bacillus suisepticus* have been elaborated by workers in Germany and America and some of them have been very widely employed. Used in conjunction with methods of immunization against swine-fever as a protection against secondary infection by the bipolar organism, these methods may be of value, but owing to the infrequency of the uncomplicated disease and its sporadic character, they are not of great practical importance.

Hæmorrhagic Septicæmia in Equines

Babes and, later, Lignières (1897) described a bipolar bacillus as the causal agent of equine influenza, and its association with that disease has been frequently confirmed. More recent investigations support the view that the *Bacillus equisepticus* is only a secondary invader, though probably responsible for the inflammatory processes in the lungs. Cases of pure hæmorrhagic septicæmia do, however, occur in equines, in which the characteristic lesions are found on post-mortem examination and the bipolar bacillus is present in large numbers in the blood and body tissues.

The *Bacillus equisepticus* (*Pasteurella equi*) possesses the general characteristics of the group, but the organism isolated by Hardenbergh and Boerner (1917) from an outbreak of hæmorrhagic septicæmia in mules failed to infect rabbits or guinea-pigs.

Symptoms.—The disease runs a very acute course, illness lasting only one or two days; the symptoms are fever, loss of appetite, restlessness or signs of colic, and occasionally subcutaneous œdema along the sternum and abdomen.

Petechiæ and hæmorrhages of various sizes are usually present throughout the body, all the serous and mucous membranes being especially affected.

Natural infection probably takes place by ingestion. Subcutaneous inoculation of culture causes a hot, painful, œdematous swelling at the site and death in one to three days.

Hæmorrhagic Septicæmia in Goats

An infectious pneumonia of goats in Turkey was shown by Nicolle and Refik Bey (1896) to be caused by a bipolar bacillus possessing the morphological and cultured characters of the hæmorrhagic septicæmia group. The disease is probably the same as that observed in South Africa, India, and elsewhere, but must be distinguished from an infectious pleuro-pneumonia of goats occurring in many European countries and India for which no causal organism has been identified.

The *symptoms* and lesions are those of acute pneumonia, with occasionally necrosis of lung tissue; shortly before death paralysis usually appears. The course of the disease is about ten days and from 70 to 80 per cent of affected animals succumb.

Hæmorrhagic Septicæmia in Elephants

Evans has reported hæmorrhagic septicæmia in elephants, and states that in Burma it is a frequent cause of death.

Clinically the disease is similar to anthrax; swellings appear on various parts of the body, the throat and tongue are swollen, and respiration is difficult. The course of the disease is very rapid and recovery rarely takes place.

Similar preventive measures to those described for cattle are applicable when dealing with outbreaks of the disease in other animals. Immune serum, by conferring an immediate protection, offers the best means of checking an outbreak, while it also possesses curative value in the treatment of animals already infected. Since it has been shown that vaccines and serum prepared from one variety of the hæmorrhagic septicæmia group of the organisms are protective against the disease in other species of animals, it is not necessary to employ a specific serum in every case, though where possible the best results would probably be obtained by so doing. Lignières' polyvalent vaccine and serum were prepared to meet this difficulty.

A. W. S.

CATTLE-PLAGUE

Contagious Bovine Typhus ; Peste Bovine (*French*) ; Rinderpest (*German*)

Definition.—An acute contagious disease chiefly affecting cattle, characterized by fever and inflammatory lesions of the mucous membranes, more particularly of the alimentary tract. All the tissues, secretions, and excretions are infective, but the specific causal agent is ultra-microscopic.

History and Geographical Distribution.—Cattle-plague has been recognized from early in the Christian era, when it spread from Central Asia into Europe. Since that time numerous epizoötics have occurred in Europe, usually on the occasion of wars in which the eastern nations have been involved.

The disease still remains enzoötic in China, Tibet, India, the steppes of southern Russia, and the Balkans; most outbreaks in other countries during recent times have occurred as the result of introduction from one or other of these areas.

Great Britain experienced serious outbreaks in 1714, 1740 to 1756, 1770, 1865 to 1867, 1872, and 1877; on each occasion the disease was stamped out by measures of destruction and disinfection, but only after great losses.

In 1881, during the first Boer War, cattle from Asia brought the disease to South Africa, but the most serious outbreak occurred there from 1896 to 1899, when over 2,000,000 cattle are said to have died. It was during this period that Koch, Theiler, Pitchford, Edington, Turner, Kolle, Danysz, and Bordet added so materially to our knowledge of methods of protective inoculation against the disease. The infection was reintroduced from German West Africa in 1901 and appeared again in 1904, but these outbreaks were speedily suppressed by the use of serum and British South African territories have since remained free from the disease. North-east Africa has repeatedly been invaded, cattle from countries where the disease is enzoötic, southern Europe and Asia, bringing the infection. In Egypt, the Soudan, and British East Africa cattle-plague continues to cause great losses. In the Philippine Islands cattle-plague first appeared in 1882; apparently it was re-introduced by Asiatic cattle for the American troops in 1898–99 and is still responsible for heavy mortality among cattle and carabaoes.

The American and Australian continents have not been infected.

Animals susceptible.—Although essentially a disease of cattle, all ruminants are susceptible and may become infected naturally. Even in cattle susceptibility varies to a great extent. The cattle of the plains of India and steppes of southern Russia possess considerable natural resistance to the disease and among them the mortality may be as low as 10 per cent, but averages in India about 40 per cent; this decreased susceptibility has probably been acquired by exposure to the disease through countless generations. In the hill tracts of India and countries where cattle-plague is not enzoötic, the susceptibility of the cattle is usually very great, and the death-rate may reach 98 per cent. Buffaloes are said to be more resistant than cattle, but recent observations in India have shown that the susceptibility of these animals varies greatly. Malay buffaloes suffer greatly, also the carabao of the Philippine Islands.

Sheep rarely show more than a mild febrile reaction when infected with cattle-plague, but in goats the disease is more severe and may cause considerable mortality.

Camels usually suffer mildly. Wild ruminants are very susceptible; during the outbreaks in Africa antelopes and buck of all kinds died in large numbers, and in India deer and black buck frequently contract the disease and succumb.

Swine are stated to possess little susceptibility, but outbreaks of the disease have been reported in these animals in Sumatra, Ceylon, and the Philippine Islands, and observations have shown that pigs can contract the disease from cattle naturally and *vice versa*. Horses, dogs, cats, rabbits, birds, and man are immune.

Etiology.—The blood and tissue fluids of affected animals, though themselves infective, contain no organisms visible with the microscope nor can any organism capable of causing the disease be obtained from such fluids by the known methods of cultivation.

Nicolle and Abil-Bey demonstrated that when diluted intestinal contents, peritoneal fluid, etc., were passed through the Berkefeld or Chamberland filter, the filtrate was infective, so that the contagion must belong to the group of ultramicroscopical organisms. Kolle found that when virulent blood is centrifugalized only the corpuscular sediment contains the virus, the supernatant fluid being non-infective; this observation has been confirmed by several workers, and Todd and White also showed by fractional centrifugalizations that the virus is chiefly present in the layer of white cells. The view that in the blood the virus is intraleucocytic was first put forward by Nicolle and Abil-Bey.

The virus is present in the blood on the first day of fever and in all the secretions and excretions very shortly afterwards.

When recovery occurs the blood ceases to be infective as soon as the body temperature returns to normal and, except in animals showing persistent diarrhœa, the excretions lose their infectivity in a few days, so that recovered animals quickly cease to be sources of infection.

As little as ·002 c.c. of blood from a sick animal taken at the height of an attack will transmit the disease by subcutaneous injection. Virulent blood applied to the mucous membranes, nose, or vagina does not usually cause infection, but nasal mucus, saliva, and fæces from sick animals almost constantly produce the disease when brought in contact with mucous surfaces. When administered by the mouth, blood and all discharges from sick animals readily transmit the disease, and no doubt natural infection most frequently occurs in this manner. It is highly improbable that the exhaled breath of sick animals contains the virus. Sheep, goats, and wild game, which suffer in varying degree from cattle-plague, may spread the disease over wide areas.

In addition to direct contact between diseased and healthy stock the disease may be distributed by means of the discharges from affected animals; thus buildings and ground become contaminated, and the infection may be carried by vermin, birds, flies, or on the boots, clothes, etc., of persons who have come in contact with the sick animals or their surroundings; the carcases of animals that have died of the disease may also be a source of infection.

Exact information regarding the length of time that the virus remains active outside the animal body under natural conditions is of great importance when framing measures for combating the disease, but the older authorities differ widely in their views on this subject. Experiments in the Philippine Islands, Egypt, and, more recently, in India have shown that the virus is not able to survive in fæces, urine, or saliva from sick cattle for a longer period than three days even when protected from sunlight; exposure to direct sunlight destroys the virus within twelve hours. Ground contaminated by animals suffering from cattle-plague does not retain the infection for more than twenty-four hours after the removal of the sick animals, and when exposed to direct sunlight infectivity is lost more rapidly. Buildings become non-infective three days after the removal of sick cattle from them.

Blood taken from an affected animal and stored under aseptic conditions may lose its virulence within three days, but under certain conditions, which are not at present satisfactorily determined, the virus may retain its vitality for much longer periods, and this has occurred even when the blood was exposed to desiccation and putrefaction. Hides lose all infectivity when dried in the sun, but in meat and bones the virus may persist for several days.

In countries where cattle-plague is enzoötic, as the plains of India and steppes of Russia, and the cattle possess considerable natural resistance, the disease spreads slowly, frequently dying out in a district after the more susceptible animals have either perished or recovered, but being reintroduced from adjoining areas when new generations of cattle have grown up. The absence of either natural or artificial barriers to its progress and unrestricted movement of stock aid in its constant maintenance. When, however, the disease is introduced into a country with highly susceptible cattle, such as England or South Africa, it assumes a highly virulent character and spreads with great rapidity. The observation has frequently been made that the virulence of the disease is greatest at the beginning of an outbreak; later the percentage of deaths declines, apparently as the result of attenuation of the virus.

During hot dry weather outbreaks are usually less severe than in cold or wet seasons of the year.

The Period of Incubation.—After inoculation of infective material into susceptible cattle the first rise in temperature occurs in from three to six days; when exposed to natural infection the period of incubation may vary from four to nine days.

Symptoms.—Considerable variation occurs in the symptoms and lesions of cattle-plague as exhibited by bovines of different parts of the world; this is more a matter of degree than of kind, but may be sufficiently marked for any one acquainted only with, say, the European form to fail to recognize the disease in Indian cattle. It may be said that none of the symptoms are constantly present in every case, but it will be sufficient to mention those breeds or localities in which the disease commonly manifests a departure from the accepted type.

For one or two days after the initial rise in temperature the animal may not show any other disturbance of health, but as the fever reaches its height, usually from 40° to 42° C. (104° to 107° F.), depression becomes marked, the head and ears droop, and the animal assumes a dull, listless attitude, with staring coat and arched back. Occasionally the depression is preceded by a short period of restlessness or, in rare cases, excitement. The appetite early becomes lessened and rumination irregular or absent, but thirst is usually increased. The respirations and pulse are accelerated, and a dry cough is frequently present during the first few days of illness but ceases as the disease develops. About the second day of fever the conjunctivæ become congested and swollen, tears flowing freely over the face; later the discharge becomes mucopurulent and viscid and dries into crusts. Catarrh of the nasal mucous membrane develops with copious discharge, at first clear but becoming purulent and fœtid as the disease progresses. The shaking of the head frequently noticed in the early stages of an attack is probably an effort to clear the nasal passages of this discharge. Twitching of the muscles of the shoulders and quarters may also occur.

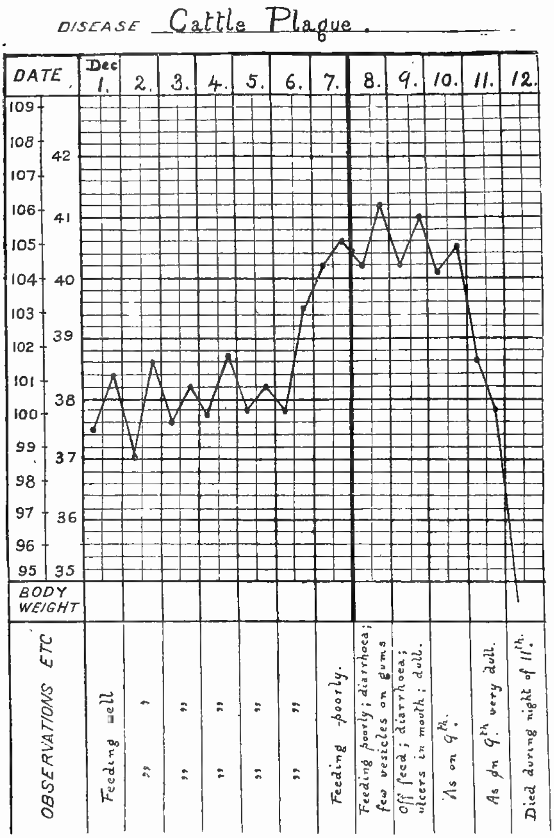

FIG. 21.—Indian hill bull, one year old, placed on infected ground on December 1st, 24 hours after removal of sick animals. Course of the disease after exposure to natural infection.

The mouth lesions of cattle-plague are usually regarded as pathognomonic, but they vary to a considerable degree in different breeds of cattle. As seen in Europe, the buccal mucous membrane becomes first reddened and later exhibits small greyish-yellow patches of thickened and softened epithelium, which spread and coalesce; the detachment of the epithelium proliferations leaves bright red erosions of the membrane. These lesions are almost constant and appear most commonly on the lips, gums, dental pad, and under surface of the tongue, but may extend to the hard palate, pharynx, and œsophagus; the epithelium of the muffle may also be shed in places. In the Philippine Islands ulceration of the mouth is said to be rare although salivation is usually present. Animals showing similar mouth lesions to those described may be met with in severe outbreaks among Indian plains cattle, but erosions are usually absent from the tongue, lips, and gums; they are more commonly present in the pharynx, which is almost invariably congested. Indian hill cattle, on the other hand, develop mouth lesions with great constancy but in the form of minute vesicles and ulcers; these show no tendency to spread, and the mucous membrane of the mouth is but slightly reddened. In South African cattle the mouth lesions usually took the form of numerous small ulcers on the reddened mucous membrane, but the diffuse desquamation of epithelium was not commonly observed. In all cases salivation is profuse and the breath has a characteristic odour.

For the first two or three days of fever the fæces are firm and glazed, but soon become soft and then fluid, mixed with mucus and having a peculiar fœtid odour; later blood may be mixed with them. There is usually straining during defæcation, but as the animal becomes weaker the fluid fæces escape involuntarily, soiling the tail and hind-quarters. Diarrhœa setting in from the fourth to the sixth day of fever is perhaps the most constant symptom of the disease. At this stage the animal shows signs of abdominal pain, grinding its teeth, looking round at its flanks, and arching its back; the inflamed rectal mucous membrane is often everted. Emaciation is rapid and weakness becomes pronounced. With the onset of diarrhœa the temperature falls rapidly, usually to subnormal before death.

The respirations are accelerated throughout an attack, and in the latter stages there is grunting, with a characteristic holding of the breath before expiration. As a result of the laboured breathing, interstitial lung emphysema frequently occurs and occasionally this leads to subcutaneous emphysema, first along the back and later spreading to the neck, shoulders, or thorax. The pulse is usually strong and rapid during the first few days of fever, but always becomes weak and thready when the temperature falls prior to a fatal termination. In cows and heifers the mucous membrane of the vulva and vagina is reddened and may show superficial erosions, with a mucous or muco-purulent discharge. Pregnant animals very frequently abort and milk secretion becomes arrested early in the attack.

Among the indigenous breeds of Eastern Europe and Asia the disease runs a milder course and in a good proportion of cases (50 to 60 per cent) ends in recovery. In these animals an eruption of epidermal scales, forming later into thick scabs, frequently occurs on the skin of the neck, shoulders, back and udder or scrotum ; this appearance is more common in hot weather and is regarded as a favourable symptom. It is rarely seen in Indian hill cattle or in the Philippine Islands, and was not observed in South Africa.

Course.—Among the more susceptible breeds of cattle the period of sickness lasts from four to eight days and usually terminates in death. When recovery occurs the temperature gradually returns to normal in a few days, but diarrhœa frequently persists for some time. In the cattle of Eastern Europe and less susceptible breeds or individuals in other countries the disease runs a mild course ; except for a febrile reaction and slight catarrh of the mucous membranes the disease might pass unnoticed. In the cattle of many countries latent piroplasmosis or trypanosomiasis may develop during or after an attack of rinderpest. Young animals always suffer more severely than adults.

Recovery from an attack of cattle-plague gives complete immunity against natural infection, usually for life. Calves born shortly after their mothers have passed through an attack of the disease possess an active immunity, but subsequent calves of such recovered animals have little increased resistance.

Diagnosis.—The recognition of cattle-plague presents little difficulty in localities where the disease is prevalent ; the first cases in a new outbreak may, however, give rise to uncertainty, when it is very important that the diagnosis should be established early and steps taken to prevent the spread of the disease. Possible sources of introduction of the infection should be carefully considered.

The symptoms to which particular attention should be paid are : a sudden marked rise of temperature, discharge from the eyes and nose, great depression and rapid emaciation, presence of erosions or ulcers in the mouth, fœtid diarrhœa or dysentery, rapid spread and high mortality.

Cattle-plague must be differentiated from the following diseases :

1. **Malignant Catarrh.**—The course of this disease is slower and it is not actively contagious ; the mucous membrane of the eyes, nose and sinuses of the head are principally involved and the typical mouth lesions are absent.

2. **Foot-and-Mouth Disease.**—This spreads more rapidly than cattle-plague but is mild and seldom fatal in adult animals. The mouth lesions take the form of large vesicles or ulcers with well-defined margins and bright red bases, and occur on the upper surface of the tongue as well as on the lips, cheeks and gums ; the nose and eyes are not affected. Vesicles also appear in the interdigital spaces of the feet.

3. **Gastro-Enteritis.**—Diarrhœa and dysentery may result from several causes, such as irritant poisons, plants (acorns), animal parasites, bacteria, coccidia (red scour), etc. The absence of symptoms or lesions other than those of the digestive tract or of evidence of contagion is sufficient to exclude the possibility of cattle-plague.

4. **Hæmorrhagic Septicæmia** and **Anthrax.**—The rapidity of death, absence of mouth lesions, and presence of the specific organism in the blood, enable these diseases to be differentiated.

5. **Piroplasmoses.**—The presence of ulcers in the abomasum and small intestine at autopsy may give rise to confusion with cattle-plague, but the absence of morbid changes of the mucous membranes of the mouth, eyes, air passages and genitals, and the history of the outbreak, are generally sufficient for differentiation. However, the existence of lesions of piroplasmosis and the discovery of piroplasmata in the blood do not exclude the possibility of simultaneous cattle-plague infection, since in countries where the piroplasmoses are widespread an attack of cattle-plague frequently causes the development of the otherwise latent infection, usually with serious results. In the same way trypanosomiasis may be found associated with cattle-plague.

Lesions.—Many of the appearances noticeable during life are to be seen on viewing the carcase of an animal that has died of cattle-plague : emaciation, sunken eyes, muco-purulent discharge from the eyes, nostrils and vagina ; hyperæmia of all the visible mucous membranes, with desquamation, erosions or ulcerations of the epithelium of the lips, gums, cheeks and under surface of the tongue ; everted anus and hind quarters soiled with fæces. The skin eruption may also be seen in the breeds of cattle in which this occurs.

The most important internal lesions are located in the alimentary tract. The mucous membrane of the soft palate and pharynx may show similar changes to those present in the mouth, or small hæmorrhages into the tissues may be present; sometimes, as in Indian hill cattle, there are minute erosions of the epithelium in the œsophagus. The rumen, reticulum and omasum are not characteristically affected; the mucous membrane may be swollen, softened, and easily detached. In the abomasum the mucous membrane is swollen and markedly congested, especially towards the pylorus and on the edges of the folds, the colour varying from bright red to dark brown or purple, with occasionally patches of hæmorrhage into the tissues. Scattered over the most affected areas are numerous small patches of necrotic epithelium which are easily detached, leaving ulcers with well-defined edges and reddened bases. Sometimes the catarrhal exudate forms a membranous deposit on the surface of the mucous membrane. The small intestine contains fluid fæces, greyish yellow to dark brown in colour, and mixed with shreds of membranous exudate; the mucosa is swollen, congested, and shows similar deposits or ulcers to those in the abomasum. Peyer's patches are prominent and covered with dirty grey deposit; extensive sloughing of the mucous membrane sometimes takes place. The alterations in the large intestine are similar but usually less pronounced; the mucous membrane at the blind end of the cæcum is very frequently inflamed. The rectum is almost constantly affected, the congestion of the mucous membrane usually appearing in broad red stripes, the so-called "zebra markings."

The upper air passages show catarrh and swelling of the mucous membrane, especially of the nose and larynx, with croupous deposits, minute hæmorrhages, or actual ulceration. The lungs are hyperæmic and sometimes contain small areas of hepatization, but the most constant lesion is an inter-lobular emphysema; air may also have escaped into the mediastinal or subcutaneous tissues. The heart is pale and flaccid; small petechiæ may be present on the endocardium; the spleen is normal. The kidneys and liver may show fatty degeneration; the gall bladder is distended and contains bile of varying colour from dark green to almost blood red; the mucous membrane may be ulcerated or necrotic in patches. The mucous membrane of the urinary bladder, uterus and vagina is usually congested and swollen. Hyperæmia of the cerebral meninges and nerve tissues may be present.

A marked leucocytosis occurs early in the attack but declines later; the blood is dark and does not coagulate readily.

Methods of Immunization.—The observation that cattle which had recovered from an attack of cattle-plague did not again contract the disease directed attention early in the eighteenth century to the possibility of producing a mild attack by artificial means. Various methods were adopted, such as the injection of nasal discharge or tears from affected cattle or of virulent blood attenuated by heat or continual passage. Little success was achieved among the susceptible cattle of Western Europe, but in Russia these inoculations were widely employed.

The occurrence of the disease in South Africa, with such disastrous results, in 1896, led to further investigations by several scientists, and various effective methods of conferring immunity were elaborated; by the energetic application of these methods cattle-plague was eradicated from the country.

1. *Immunization by means of Bile.*—The Boers in South Africa appear to have been the first to use the bile of animals dead from cattle-plague as a means of giving protection. Koch in 1897 experimented largely with bile, and demonstrated that a valuable degree of immunity could be produced by its injection into susceptible cattle. Koch's method consists in the inoculation subcutaneously of 10 c.c. of bile taken from an animal killed between the sixth and eighth day of the disease. Hutcheon recommends bile to be taken on the tenth day after infection, or the fourth or fifth day of visible sickness. Usually the animals used for the production of bile have to be given cattle-plague artificially, and this necessitates the sacrifice of from 4 to 7 per cent of the herd; one animal yields enough bile to inoculate 20-25 cattle, but in about one out of every five the bile is unsuitable. It was the opinion in South Africa that animals infected naturally or by the digestive tract gave more bile, with stronger immunizing properties than those injected subcutaneously with virulent blood; hence it was the practice to infect cattle intended for bile production by smearing the muzzle with fæces or nasal discharge from a sick animal.

Koch advised that only biles that were clear, dark green in colour, odourless and giving a white froth on shaking, should be employed, but experience showed that it was only necessary to reject those of a light yellow colour or which contained blood, provided the material was clear and free from any putrid smell; if possible, bile from several cases should be mixed together to secure uniform results. The bile injection usually causes no constitutional disturbance, but immunity seldom becomes established before the tenth day. The protection given is then sufficient to resist either natural infection or the injection of virulent blood, and lasts on an average about four months (Haedicke). The duration of the immunity is, however, very variable even in a single herd, the limits having been placed at between four weeks and six months (Edgar), so that it is impossible to know the degree of resistance possessed by the animals of a herd two or three months after bile inoculation. This disadvantage was experienced in South Africa, when it was necessary to regulate the dose of serum for herds previously treated with bile. Larger doses than 10 c.c. do not appear to give longer immunity, but the bile injection may be repeated every three months to maintain protection (Theiler); this course was recommended in South Africa.

Fresh bile from an animal that has died of cattle-plague contains the virus of the disease and also antibodies which restrain its activities when injected into a susceptible animal (Koch). It is the presence of the organism, whether modified (Turner) or unmodified (Koch), that enables the bile to produce an "active" immunity, but in some cases it is also responsible for an actual attack of the disease, especially in very susceptible animals (Hutcheon), thereby setting up fresh centres of infection. Bile taken early in an attack of the disease is very likely to cause infection, as sufficient time has not elapsed for the formation of specific antibodies (Kollel). The risk of producing the disease is lessened by storing the bile for a day or two, but after that time the virus is destroyed and the antibodies remaining give only a "passive" immunity of short duration; the bile also quickly undergoes putrefaction.

To prevent any possibility of conveying the disease by bile inoculation Edington added one part of glycerine to two parts of bile, and stored the mixture for eight days before use, but in practice two days' storage was found to be sufficient; the dose is from 15 to 35 c.c. injected subcutaneously. With the destruction of the virus the power of producing an active and lasting immunity is lost, so that the protection given is of short duration and depends solely on the antibodies present in the bile. Glycerinated bile is said to have strong curative properties in doses of 50 to 100 c.c. intravenously (Hutcheon), and will keep effective for a year.

To produce an active immunity the bile and glycerine inoculation is followed ten days later by an injection of 0·2 c.c. of virulent blood, and after a further interval a larger dose of virulent blood is frequently given. By this means most of the animals are passed through a mild attack of the disease and recover. This method gave good results in some countries, but in South Africa, where over 390,000 animals were inoculated with a loss of only 8·24 per cent, the injection of virulent blood was discontinued on account of the uncertainty of the reaction produced and the risk of communicating other diseases; glycerinated bile was mainly used alone to give a passive immunity until the infection had died out (Hutcheon).

Rogers recommended passing the bile through a porcelain filter, the virus of the disease and any putrefactive organisms that might be present being thus removed. The filtrate therefore has the same effect as glycerinated bile, but a larger proportion of bile can be utilized.

2. *Immunization by means of Blood Serum.*—Semmer in 1893 found that serum from cattle which had recovered from cattle-plague was able to protect susceptible animals when injected in doses of 100 c.c. or more, but this protection was only of short duration. Koch confirmed this observation early in 1897, and attempted to produce an active immunity by adding a small amount of virulent blood to the serum and injecting both together. Most of the early experimental work in connection with anti-cattle-plague serum was, however, carried out by Pitchford and Theiler, Danysz and Bordet and Turner and Kolle, all of whom aimed to increase the potency of the serum and to apply it for the production of both passive and active immunity in cattle, according as it was used alone or in conjunction with the disease contagion.

The Preparation of Anti-Cattle-Plague Serum

The method adopted by Turner and Kolle was to inject 100 c.c. of virulent blood into an animal shortly after its recovery from an attack of the disease, natural or artificially produced; this was followed by injections of 200, 500 and 1000 c.c., time being allowed between each injection for the resulting reaction to subside. After the last injection the animal was bled three times for serum at intervals of about ten days and then reinjected with 2000 c.c. virulent blood; three bleedings were taken after each injection, these being increased according to the size of the animal up to 5000 c.c. In this way a serum of high protective value was obtained. Later Nicolle and Abil-Bey (1899) showed that animals which had just recovered from an attack of the disease could at once tolerate a large injection of virulent blood and produce a strong serum, thus greatly shortening the period of hyper-immunization.

During recent years the technique of serum manufacture has been greatly improved, chiefly by workers in Egypt, India and the Philippine Islands. The method adopted at the Imperial Bacteriological Laboratory, Muktesar, India, where the issues average 1000 litres of serum a month, is briefly as follows:

Both plains buffaloes and hill cattle are employed for serum production and to furnish virulent blood for hyper-immunization, but only the latter, being highly susceptible, are used for maintaining the virus in an active condition. Plains cattle were found to produce serum of low potency and are not now employed. The serum-making animals are first given 1 c.c. of virulent blood and a dose of serum, according to the simultaneous method of inoculation described later, by which they are made to pass through an attack of cattle-plague. At the height of the reaction, sixth or seventh day, they are bled, about 4 c.c. to the lb. body-weight, and the blood used for hyper-immunizing other animals (Holmes). By the twelfth or fourteenth day the animals have recovered from the reaction, and on the twentieth or twenty-first day they are bled once (6 c.c. per lb.) for serum; this serum is only slightly less potent than that from hyper-immune animals (Holmes). Three or four days later they receive an injection subcutaneously of virulent blood and an equal volume of 0·5 per cent potassium citrate solution, at the rate of 5 c.c. of the mixture per lb. body-weight; a slight thermal reaction lasting three or four days usually follows this injection. On the tenth day blood is taken at the rate of 6 c.c. per lb. and again on the fourteenth day at the rate of 8 c.c. per lb.; or three bleedings of 6 c.c. per lb. are taken at intervals of four days. After a short rest the animals are reinjected with virulent blood and citrate solution and the bleedings repeated as before; the dose of blood and citrate mixture is increased after each series of bleedings until 8 c.c. per lb. is injected, after which it remains constant.

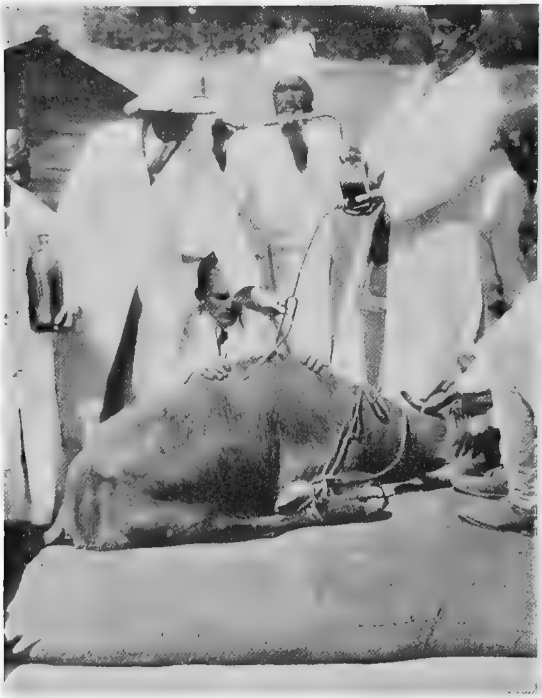

Fig. 22.—Preparation of anti-cattle-plague serum. Injection of virulent blood into serum-making buffalo.

Hill cattle for the supply of virus are inoculated with virulent blood and bled (4 or 5 c.c. per lb.) at the height of the attack, usually on the fifth day; on the following day they are bled to death, a litre of salt solution being injected into the jugular vein shortly before death to wash out the vessels. The blood is mixed immediately with an equal volume of sterile potassium citrate solution (0·5 per cent), which prevents clotting and causes hæmolysis of the red cells; the mixture is more rapidly absorbed after injection than defibrinated blood, and abscess formation is less common.

When buffaloes are bled for serum the blood is drawn into potassium oxalate solution, which prevents clotting and allows the red corpuscles to sediment; this they do rapidly. After standing eighteen hours the plasma is drawn off, calcium chloride solution added, and the resulting clot compressed by means of weights; the clear serum is decanted and 0·5 per cent carbolic acid added. The blood from hill cattle after oxalating or defibrinating must be centrifugalized to separate the serum from the corpuscles, since the latter do not settle of their own accord as in buffalo blood.

When from 600 to 800 litres of serum have been prepared, from both hill cattle and buffaloes, the whole is mixed and tested on susceptible hill cattle in varying doses against a simultaneous injection of virulent blood; the amounts of serum given are 27, 54 and 81 c.c. per 600 lb. body-weight. Serum which protects at 54 c.c. per 600 lb. is issued for use; if below this standard a proportion of first bleeding serum is added and the whole re-tested. Lingard estimated that hill cattle required eighteen times as large a dose of serum to give them protection as that necessary for plains cattle of lowest susceptibility; the dose of serum for the latter would therefore be only 3 c.c. per 600 lb., but to cover variations in resistance, from 5 to 15 c.c. are usually given.

As large numbers of animals have to be slaughtered to supply virulent blood for hyper-immunization various methods of increasing the yield of virulent material have been suggested. Nicolle and Abil-Bey found that the peritoneal fluid could be utilized, and Ruediger injected large amounts of potassium citrate solution into the peritoneal cavity shortly before bleeding the infected animals to death, after which the fluid was collected and mixed with the virulent blood for hyper-immunization. By injecting saline solution intravenously during the final bleeding a larger proportion of the blood in the body can be obtained.

The protective properties of anti-cattle-plague serum may be applied in various ways according to circumstances for the immunization of animals, the two chief methods being the "serum alone" and "serum simultaneous."

(*a*) *The Serum Alone Method.*—This simply consists in the subcutaneous injection of a suitable dose of anti-cattle-plague serum into the susceptible animals it is desired to protect. No disturbance of health is caused by the inoculation, but a passive immunity is at once established, the duration of which varies according to the dose and potency of the serum given and the susceptibility of the individual or breed treated. The dose of serum employed in South Africa during the earlier inoculations was 20 to 30 c.c. per 600 lb. body-weight,[1] but later much larger doses, up to 200 c.c., were found to be necessary. Experience in the Philippine Islands and India with the most susceptible breeds of cattle has shown that doses of 90 to 150 c.c. per 600 lb. body-weight must be used to produce immunity; Indian plains cattle, however, only require from 5 to 20 c.c. per 600 lb. to protect them against a fatal attack of the disease. The older authorities state that serum can confer immunity for periods up to four months, but later experience has proved that even after relatively large doses protection begins to decline in five or six weeks and a standard dose cannot be relied upon to protect for more than ten to fifteen days.

In countries where cattle-plague is epizoötic and systematic sanitary police measures aiming at the eradication of the disease are in force, serum may be employed to give absolute immunity to all susceptible animals in the affected district, thus preventing the spread of the infection. But since the immunity must be maintained as long as the disease exists it is necessary to repeat the serum inoculations at intervals of ten to fifteen days until the sick animals have either died or recovered and all risk of reinfection has disappeared. This was the method by which cattle-plague was finally eradicated in South Africa.

When, however, the disease is enzoötic in a country and effective quarantine measures cannot be maintained, a position of affairs existing in India, then the purpose of the serum inoculation is to enable as large a number of animals as possible to pass safely through an attack of the disease and thus acquire an active and lasting immunity. Accordingly the in-contact animals at an outbreak are given a suitable dose of serum and deliberately mixed with the sick in order that they may become infected before the temporary protection given by the serum has passed away. Since this procedure tends to maintain the disease it should only be carried out when dealing with herds already infected and when the more reliable serum simultaneous method of producing permanent immunity cannot be applied. The curative value of serum is not marked unless it can be given early in the disease; a single large dose injected intravenously before the third day of fever will usually modify the attack and enable recovery to take place.

Carbolized serum stored at a low temperature will retain its full potency for a year, and even after several years may still have considerable protective value.

The chief advantages of the use of serum alone are: (1) Immunity is conferred immediately and animals in the incubative period of the disease may be saved; hence serum is especially valuable for treating infected herds. (2) The injection is harmless; pregnant animals and those in milk are protected without risk; neither cattle-plague nor any other disease can be produced by the serum. (3) The serum can be prepared and stored ready for use.

The great disadvantage of the use of serum alone is the limited period for which immunity is conferred.

In South Africa before large supplies of highly potent serum became available the defibrinated blood of recovered ("salted") cattle was used in the same way as serum alone. By injecting the salted cattle subcutaneously with large doses of blood from sick animals the potency of their blood is considerably increased and two or three bleedings can be taken from them at intervals after each injection of virulent blood, in the same manner as described for the preparation of anti-cattle-plague serum.

The usual practice was to take the salted cattle to the scene of the outbreak; they were then bled by inserting a cannula into the jugular vein and drawing the blood direct into a clean sterilized bucket. The blood must be whisked briskly with a wire brush for about ten minutes to remove the fibrin and prevent clotting; after straining through sterile muslin the blood is ready for injection. This method may be employed in case of emergency when anti-cattle-plague serum is not available.

The disadvantages of defibrinated blood as compared with serum are that the dose is about double, owing to the presence of the blood-cells. It must be used within a few hours of being taken, as it quickly becomes septic, and in countries where blood infections such as piroplasmosis and trypanosomiasis exist there is a danger of transmitting these diseases in the defibrinated blood. In South Africa fresh defibrinated blood was believed to be more effective in treating sick animals than serum (Danysz and Bordet).

(*b*) *Serum Simultaneous Method.*—The short duration of the immunity conferred by serum alone led several investigators to try methods of combining with it the virus of cattle-plague so as to produce a mild attack of the disease and thus an active and lasting immunity.

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[1] The weight of cattle may be calculated with sufficient accuracy by measurement; $G^2 \times L \div 300 =$ weight in pounds, where G=girth behind the elbow in inches and L=length from point of shoulder to point of buttock in inches.

In South Africa Pitchford and Theiler (December 1896) and Bordet and Danysz (1897) introduced the method of injecting animals with defibrinated blood or serum from recovered or hyper-immune cattle and then rubbing infectious saliva or fæces into their nostrils; a good proportion of reactions may thus be obtained.

The method as it is now practised was first worked out by Turner and Kolle (July 1897), and consists in the injection subcutaneously of a small amount of virulent cattle-plague blood on one side of the animal and on the other a suitable dose of anti-serum; by using standardized serum and ascertaining the correct dose according to weight for cattle of different breeds or countries, a large percentage of the animals treated may be safely passed through an attack of the disease and given permanent immunity. The virulent blood is taken from a sick animal at the height of the disease and the quantity injected is usually 0·5 or 1 c.c. If it is necessary to transport the blood to a distance or store it for several days, it should be taken with aseptic precautions from a highly susceptible animal early in an attack of cattle-plague and kept in sealed tubes at a low temperature; it will then remain virulent for a considerable time. Sheep inoculated with virulent blood may also be utilized for conveying the virus.

The dose of serum must be carefully determined for each class of animal; if too small, severe, and possibly fatal, attacks will follow, while large doses of serum may prevent any reaction from taking place. Edington, Nicolle and others state that if no reaction occurs the immunity conferred is of short duration, being due entirely to the serum injected; more recent observations have shown, however, that an active immunity lasting for several months may be produced even when no reaction follows the double inoculation.

In India the following scale of doses of serum per 600 lb. body-weight have been found safe but to allow of a good proportion reactions: pure bred imported cattle, 150 to 200 c.c.; cross-bred imported and Indian plains cattle, 90 to 120 c.c.; Indian plains cattle, 10 to 20 c.c.; hill cattle, 90 c.c.; buffaloes, 20 to 40 c.c.

In South Africa Turner and Kolle claimed that they obtained reactions in 90 per cent of cattle with a loss of only 1½ per cent. Hutcheon, however, found the proportion of reactions unsatisfactory and adopted the practice of giving the virulent blood injection forty-eight hours before the serum, but a further injection of serum was frequently necessary to control the reaction. Rogers proposed the re-inoculation of all animals failing to react with 10 c.c. of virulent blood ten days after the simultaneous injections. This method has given good results in practice; it increases the safety of the inoculation by allowing the use of larger doses of serum, and even if no reaction follows the second injection of virulent blood the immunity is strengthened.

In spite of every care in regulating the dose of serum a certain number of individuals will be encountered with abnormal susceptibility, and these will suffer from severe and sometimes fatal attacks of cattle-plague. This variation in susceptibility is greatest in those countries where cattle-plague has previously existed or is enzoötic; the cattle of areas newly invaded have usually a fairly uniform high susceptibility. Whenever possible, tests on a small number of animals of each breed or class should be carried out to ascertain the correct dose of serum before commencing inoculations on a large scale.

All the reacting animals are capable of spreading the disease so that the method is most applicable to infected herds or under conditions of rigid isolation. Hutcheon states that in South Africa the danger of infecting cattle in the neighbourhood was found to be very slight.

Animals advanced in pregnancy and milking cows should be given large doses of serum only to protect them from infection, as a severe reaction is likely to induce abortion and diminish the milk yield in such cases.

In countries where piroplasmosis (red water) and trypanosomiasis (surra, nagana, etc.) exist, the danger of conveying these diseases by the virulent blood injection must be guarded against; microscopical examination of the blood before injection should always be carried out. When piroplasmosis infection occurs the symptoms usually develop directly after the rinderpest reaction and may be treated by the injection of trypanblue (150 to 200 c.c. of a 1 per cent solution subcutaneously).

Other diseases, as foot-and-mouth disease and heartwater, may also be transmitted by the virulent blood inoculation.

The chief advantages of the serum simultaneous method are: (1) Protection from natural infection is conferred immediately and animals already infected may be saved. (2) Permanent immunity is established in all cases giving good reactions, and even those failing to react are protected for a considerable time, usually long enough to carry them through the particular outbreak. (3) As compared with the bile and virulent blood methods, reactions are more reliable and under better control and the injections are made at one time.

The disadvantages of the method are the same as those of the other methods of conferring permanent immunity: (1) The risk of spreading the disease, though experience has shown this to be slight. (2) A small percentage of losses from the reactions. (3) The risk of conveying other diseases by the virulent blood inoculation.

Among Hindu races the injection of blood and slight risk to the animals is objected to on religious grounds, so that the method cannot be applied to Indian-owned cattle.

Treatment.—Medicinal agents appear to have little or no effect on the course of the disease; the internal administration of carbolic acid (one drachm), potassium permanganate (two to four drachms), or iodine has been recommended, but the results are uncertain. Astringents given for the purpose of checking the diarrhœa are distinctly harmful. Anti-cattle-plague serum in large doses exerts an undoubted effect if injected, preferably intravenously, before the onset of marked clinical symptoms; fresh defibrinated blood from a salted animal may also be employed. When possible, nursing and suitable diet are of great assistance in aiding recovery. Warm clothing, shelter from rain or sun, green food and rice gruel should be provided. Before the onset of diarrhœa plenty of water, with Epsom salts, two ounces twice daily, may be allowed; but when purging commences the amount of water should be reduced and thin rice gruel substituted; stimulants (alcohol, nux vomica, ginger) may be administered.

As all the discharges of the sick animals are infective, strict isolation and disinfection of premises and attendants must be maintained.

Measures of Control and Eradication.—In the British Isles the introduction of cattle-plague is prevented under the Diseases of Animals Act, 1894, by allowing the importation of live stock from those countries only which are free from the disease; all cattle imported must be slaughtered at the port of landing. Should the disease gain entrance to the country, however, it would be dealt with, under the Cattle-Plague Order, 1895, by immediate slaughter of all infected and in-contact animals and the rigid measures of quarantine and disinfection prescribed.

Most other countries free from the disease enforce quarantine regulations against those in which cattle-plague prevails; the restrictions extend to fresh hides and other animal products as well as live stock, but dried and salted hides, hair, wool, etc., are not regarded as dangerous.

When cattle-plague invades a country and becomes widely spread so that methods of extinction by slaughter are impracticable and the animals are likely to be exposed to risk of infection for a long period, it is necessary to adopt one of the methods of conferring active immunity against the disease. These are: (*a*) inoculation with pure bile; (*b*) inoculation with glycerinated bile followed by pure bile or virulent blood; (*c*) serum simultaneous method.

If standardized serum is obtainable the simultaneous method is the most reliable for giving permanent protection to the animals. It was successfully employed in South Africa, and is widely practised in the Philippine Islands and Egypt.

As soon as the disease is under control and quarantine measures can be enforced, or when dealing with isolated sporadic outbreaks, the use of virulent blood should be prohibited as tending to maintain the disease, and only serum alone, defibrinated immune blood or glycerinated bile employed; serum should be used when available, but otherwise cattle owners themselves can prepare the defibrinated blood or glycerinated bile and carry out the inoculations. Animals already infected may be given large doses intravenously to aid recovery, while all in-contacts and clean herds in the neighbourhood should receive injections at short intervals, as long as the disease exists, in order that an immune zone may be maintained round the infected herd. All movements of cattle within this area must be prevented, attendants subjected to frequent disinfection, carcases and discharges of sick animals burnt, and all infected premises thoroughly disinfected.

Pure fresh bile should not be employed in infected herds if other methods are available, as it tends to increase the virulence of the disease in animals already infected, and, owing to the interval which elapses before immunity is established, it may fail to protect many of the healthy animals from infection. It may be injected eight or ten days after serum or glycerinated bile to give more lasting immunity.

A period of thirty days should be allowed after the death or recovery of the last case of cattle-plague before a herd is declared free from infection.

When cattle-plague is enzoötic and there is the constant risk of infection, the cattle should be permanently immunized by the serum simultaneous method; as the calves do not inherit immunity from their mothers it is necessary to treat the young stock periodically to maintain a protected herd. In India the serum alone method is the only one acceptable to Hindu cattle-owners, but by mixing the inoculated and sick animals together a considerable proportion acquire lasting immunity.

Cattle-Plague in Sheep and Goats.—Several extensive outbreaks of cattle-plague among sheep and goats have been reported in Europe, and these animals frequently become infected during epizoötics of the disease in bovines. As the symptoms are usually less pronounced than in cattle, the existence of cattle-plague in the flock may be overlooked and the infected animals become responsible for spreading the disease to clean areas.

The *symptoms* and anatomical changes resemble those seen in cattle, and comprise fever, discharge of tears, nasal catarrh, great depression, first constipation and later fœtid diarrhœa, and inflammation of the mucous membrane of the digestive tract and air passages. Sheep rarely show ulcerations of the mucous membranes, but pneumonia is a frequent complication. Erosion ulcers and yellowish epithelial deposits are usually found on the mucous membrane of the mouth and abomasum in the case of goats. The affected flocks may be treated by serum injections (5 to 10 c.c.), and similar quarantine measures to those employed for cattle should be adopted.

A. W. S.

CONTAGIOUS BOVINE PLEURO-PNEUMONIA

(Pleuro-pneumonia contagiosa bovum)

La Peripneumonie Contagieuse (*French*) ;
Lungenseuche der Rinder (*German*)

Definition.—A specific contagious disease of cattle with lesions mainly confined to the lungs and pleura and characterized by a marked exudation of sero-fibrinous lymph into the interstitial connective tissue of these organs.

History.—Contagious bovine pleuro-pneumonia is an old disease which was recognized as a serious affection towards the end of the seventeenth century. In the middle of the eighteenth century there was a great epidemic among cattle in Switzerland, and the disease was studied by Bourgelat and Haller. From Switzerland it spread to France and Germany, and then about 1820 it invaded Holland and Belgium. Great Britain was infected by the importation of Dutch cattle in 1841 ; British stock transmitted it to Sweden and Denmark in 1843 ; Cape Colony became infected by means of imported Dutch cattle in 1854 ; and the United States and Australia were each infected by means of imported British cattle in 1859.

Meantime Willems, a Belgian doctor, suggested a method of prevention by means of vaccination with the sero-fibrinous exudate from the lungs, which method, whilst vigorously criticised at first, still survives as an important practical method of immunization wherever pleuro-pneumonia exists.

Throughout the greater part of the nineteenth century pleuro-pneumonia prevailed over a great part of Europe, including Great Britain, but by 1910 the disease had been successfully eradicated from European countries with the exception of Russia and Spain. The United States became free from the disease in 1892 ; the last cases occurred in Great Britain in 1898 ; but the disease still remains in Australia, parts of Africa and in Asia.

Next to Cattle-plague (Rinderpest) pleuro-pneumonia is the most serious contagious disease of cattle, and some idea of the ravages produced by it may be obtained by a study of the losses which resulted in Great Britain during epidemics of the disease. In 1860, 187,000 animals, of a value of 19 million pounds sterling, died as a result of the disease. From 1870 to 1890, 63,333 affected cattle were slaughtered and 7110 died. Similar huge losses have resulted in other countries affected, and pleuro-pneumonia still takes a heavy toll in some of the Australian States.

Etiology.—From the time of Willems, who described the presence of corpuscles exhibiting Brownian movement in the serous exudate from the lungs, and who first demonstrated experimentally the infective properties of this lymph, many investigators have sought to determine the specific cause of the disease. Pasteur worked at the problem in 1882 and was able to affirm that the virus was a specific one, the same in all cases of the disease ; that it was not associated with visible bacteria ; and that it could not be cultivated in ordinary nutrient broth.

Continuing the savant's researches Nocard and Roux were able by an ingenious method to obtain pure cultures of the virus in 1898, and it is to them and their collaborators that we owe our knowledge of the habits and properties of the virus. The method consisted in the inoculation of nutrient broth with a few drops of lung lymph from an affected animal, the broth being contained in small collodion ampoules, which were then introduced into the peritoneal cavity of rabbits. Osmosis takes place freely under these circumstances and the organism is thus supplied with nutriment whilst being protected against the action of leucocytes. After fourteen days the rabbits were killed, and the collodion sacs, by this time fixed by means of plastic adhesions, were removed. The broth was found to be slightly hazy or opalescent, and on microscopic examination was found to contain numerous small refractile bodies appearing as mere points and so minute that the highest magnifications were insufficient to determine any definite form.

Control sacs of broth similarly treated in every way, except that they were not inoculated with pleuro-pneumonial lymph, remained clear and sterile. From the infected tubes other ampoules could be inoculated and the organism grown by repeating the same procedure. By this means pleuro-pneumonia was shown to be due to an extremely minute micro-organism so small as to be about the limit of visibility, and able to pass through a Berkefeld V or a Chamberland F filter. These filters are proof against ordinary bacteria, but they will allow the passage of the ultra-visible or filtrable viruses, among which are found the causal viruses of a number of diseases, notably " foot-and-mouth " disease and " sheep-pox."

The infective material, whether blood or other fluid obtained from the lesions of the disease, usually contains a high percentage of albumin, and in a fluid so viscous the active virus is unable to pass through the fine pores of a bacterial filter. To allow of its passage the fluid must be diluted with sterile water or nutrient broth, in the case of pleuro-pneumonia until there is only 2 per cent of the lymph in the mixture. When this is done the virus passes through the filter, whilst at the same time any contaminating bacteria are held back, and the filtrate will contain only the active pleuro-pneumonia virus. Ordinary nutrient broth is not a suitable medium for the cultivation of the pleuro-pneumonia virus, and instead a special broth known as Martin's broth, to which has been added 8 per cent of ox serum, has to be used. The following procedure is advised by Dujardin-Beaumetz for obtaining the virus in pure culture from a natural case of pleuro-pneumonia. A small quantity of the characteristic lung lymph or pleural exudate is taken from a recently dead animal, and added to sterile Martin's broth to the extent of 2 per cent. The mixture is then filtered through a Berkefeld V, or Chamberland F filter, and when this is complete, enough ox serum to form 8 per cent of the total is passed through the filter and mixed with the filtrate. The filtrate is now incubated in test-tubes at 37° C. After three or four days' incubation the medium in the tubes is seen to be slightly hazy or opalescent, but the change is so slight that control tubes containing Martin's broth and serum but no lung lymph (and therefore remaining sterile) have to be incubated simultaneously and, themselves remaining quite clear, used to show up the opalescence of the tubes containing the pure culture of living virus.

From the pure culture obtained in this way, serum agar tubes made with Martin's broth may be sown and a growth on a solid medium obtained after three or four days. The colonies are small and transparent, like drops of dew. On the fifth day the centre becomes brownish and more or less opaque. The growth is only slightly raised from the surface, but rather grows down into the medium.

Microscopically with a magnification of 1500 to 2000 diameters the organism in broth cultures appears in the form of minute refractile points, short spirillæ, and asteroid bodies. Borrel by special staining methods, such as Lœffler's stain for flagella, has determined the pleomorphic characters of the organism, and because of its branching mycelia and the presence of a capsule has suggested the name *Asterococcus mycoides.* The organism stains with the ordinary aniline stains, better with carbolfuchsin, and with Giemsa's stain. It is Gram negative. Examination by the hanging drop method proves it to be non-motile and therefore not a spirillum or spirochæte.

Cultivation.—The organism is an aërobe, growing best at a temperature of 37°-38° C. The virus is easily destroyed by heat, for a temperature of 58° C. is fatal to it. No spores are formed. In ordinary culture tubes the organism dies out after about a month in the incubator, but in sealed tubes kept at a temperature of 10°-15° C. it remains active for ten months or more (Dujardin-Beaumetz). The serum from a piece of frozen lung is found still virulent after more than a year. Glycerine, and carbolic acid to the extent of 0·5 per cent, are used to preserve the virus or lymph for immunizing purposes and have little or no effect on its virulence.

Pathogenicity.—The only animals commonly affected naturally are cattle, but buffalo, camels and deer are also susceptible. None of the experimental laboratory animals can be infected, and the disease is not transmissible to man. Among cattle a proportion approaching 20 per cent appear to be immune to natural infection, and further, calves under 2 months old are much less susceptible to infection, whether natural or artificial, than older animals. Stalled or stabled cattle appear more susceptible than those on open range.

Mode of Infection.—Experimentally by subcutaneous injection of a few drops of pure culture in bouillon just behind the elbow a swelling is produced in from six to twenty days. This is hot, painful and tense, and commencing as a slight elevation the size of the palm of the hand, with the point of inoculation as the centre, it gradually enlarges to a flattened tumour of 12 inches or more in diameter with a sympathetic œdema surrounding it and extending along that side of the body. Meantime the animal shows constitutional symptoms. There is loss of appetite, depression and fever. There is also considerable swelling and tenderness of the prescapular axillary and precrural lymphatic glands of that side. The animal usually succumbs at this stage, and it is often found on post-mortem examination that the intercostal muscles are involved and there may even be some exudate into the pleural cavity.

Exactly the same phenomena occur if fresh lung lymph taken from an animal recently dead of the disease is used. Indeed, this experiment was first performed, and the result described, by Willems in 1852 in his attempt to determine the cause of the disease.

On incising the swelling produced by inoculation in this way a striking resemblance to the lung lesion produced in a natural attack is at once evident. The subcutaneous tissue is infiltrated with a very abundant exudate of clear straw-coloured lymph and the strands of connective tissue appear like the veins in marble. In muscle the connective tissue between the bundles of fibres is similarly infiltrated and the likeness to pleuro-pneumonia lung is very marked. At the same time, it is remarkable that the natural disease, with lung lesions and the power of infectivity, is not set up either by means of subcutaneous injection of lymph or by any other experimental procedure. The reaction to the experimentally produced lesions does, however, confer an active immunity both against natural infection with the disease and against experimental infection.[1] Since subcutaneous injection of virulent lymph anywhere about the trunk usually causes a fatal illness the method is inapplicable as a practical method for producing immunity. Willems suggested and practised the method of vaccination or inoculation of virulent lymph under the skin of the tail near the tip. A few drops of lung lymph or pure broth culture of the virus when injected subcutaneously near the tip of the tail cause a swelling about 2 inches long, which appears in from six to twenty-two days, the part becoming somewhat hot and tender but not to a serious degree. If the swelling is much larger than this, say 4 inches long, the reaction is too severe for safety. The tail is then carried stiffly, the skin becomes rough and harsh to the touch, and if left alone either the tail will become gangrenous and slough off, or, more serious, the swelling may extend rapidly upwards and general infection occur. Sloughing of the tail occurs in from 5 to 15 per cent of cases. The number of cases of general infection is much less. In such cases there is a painful swelling in the perineal region, and post mortem it is discovered that the serous exudate has been poured out into the connective-tissue spaces inside the pelvis, the lymphatic glands of the part are greatly enlarged and œdematous, and general lymphatic infection followed by death of the animal is the result. Many of these cases can be prevented by early amputation of the tail when the reaction is seen to be too severe. In quite young calves of 1 to 2 months old there is often no reaction. In other cases calves may suffer from a multiple arthritis as the result of inoculation.

Nocard's experiments at the Alfort Veterinary School showed that intravenous injection of virulent lymph failed to infect, and as a consequence produced no immunity. This result points to the unsuitability of the blood for the growth of the micro-organism, and in agreement with this it is found that the blood of an infected animal is not virulent. Further, intrapulmonary injection of pleuro-pneumonia lymph failed to produce any visible reaction although immunity resulted. On the other hand, the injection of lymph into the pleural cavity sets up a severe pleurisy with abundant effusion of infective lymph, and with many plastic adhesions and infiltration of the thoracic lymphatic glands. Nocard was unable to produce either reaction or immunity by drenching cattle with infected lymph or cultures, and the spraying of cultures of the micro-organism in front of the nostrils of healthy cattle failed to infect.

How natural infection occurs is not fully understood, but certain facts of experience can be stated. All observers with much experience of pleuro-pneumonia are agreed that direct contact of diseased cattle with healthy animals is the most certain method of infection. There is no evidence to suggest that the virus of pleuro-pneumonia can live and propagate naturally outside the animal body. It is frequently stated that the virus may remain active and infective for several months in cow-sheds and stables where infected animals have been. Soiled fodder and litter, and attendants are also stated to be able to convey the infection. On the other hand, officers of wide experience in the control of pleuro-pneumonia confidently affirm that cowsheds, etc., never convey the disease. One such authority says: "We feel quite safe even if healthy animals should enter or remain in paddocks or sheds immediately after affected animals have been killed or removed."[1] The great majority of outbreaks can, however, be traced to the presence of an affected animal. Such an animal may have been retained after a previous outbreak either as a recovered animal or as one which it was thought had escaped infection, having shown little or no sign of illness. Even more commonly the infecting animal is one which has been recently purchased, being in fact a "recovered" animal sold by some unscrupulous or ignorant owner.

Affected animals appear to be infective at any stage of the disease, although only rarely in the incubative period before recognizable pleuro-pneumonia symptoms are to be seen. Affected lungs retain the active virus for many months. Walley, from his large experience of the disease in Great Britain, affirmed that the virus remained active in encapsuled lung lesions for a period of fifteen months. Other authors have put the length of time much greater. Lung lymph from a hepatized lung frozen and kept in cold storage was shown by Laquarrière to be virulent after more than twelve months, and Dujardin-Beaumetz was able to keep pure culture virulent and active for over ten months at a temperature of 10°-15° C. in sealed ampoules.

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[1] See Immunization, p. 128.

[1] W. A. N. Robertson, B.V.Sc., Chief Veterinary Officer, Victoria, to whom I am indebted for valued assistance.—H. A. W.

The medium of infection in natural cases is most probably inhaled air from an affected animal. Cheauveau showed that an animal in the acute stages of the disease could transmit it to a healthy one when the animals were made to breathe out of opposite ends of the same sack passed over the heads of them both.

Australian experience at the present time fully supports older British experience that old "lungers," *i.e.* recovered (?) animals with encapsuled or necrotic foci in their lungs, are the greatest sources of danger and the chief factor in causing new outbreaks.

Symptoms and Course of the Disease.—The period of incubation in experimental cases, after subcutaneous injection of the virus or pure culture of the organism, varies from six to twenty days prior to the rise of temperature pointing to infection. In natural cases it appears to be somewhere about fourteen days up to the initial rise of temperature.

In an animal the subject of a natural attack there are at first some rather indefinite signs of ill-health. The appetite is capricious, rumination is more or less suspended, the animal restless, preferring to isolate itself from the herd, and the coat is a bit harsh. In a cow the milk secretion often falls off. If the temperature is taken at this stage it will almost certainly be raised—to 104° F. or more—and the respiration is somewhat accelerated. After several days the animal is likely to develop a cough, especially noticeable on rising or when coming to rest after being driven or made to move quickly.

From this time the condition usually gets rapidly worse until all the signs of pneumonia and pleurisy are evident. The animal prefers to stand quietly, with elbows turned out and chest fixed so that most of the work of breathing may come upon the diaphragm and abdominal muscles—so relieving the inflamed and painful pleura. Pressure over the chest-wall causes pain, and may elicit a grunt; indeed a grunt often accompanies each expiratory act. Percussion of the chest-wall is objected to because of the pain it causes, but if persevered in will reveal the onset of hepatization of lung areas, and the accumulation of fluid in one or both sides of the chest up to a certain definite level. Auscultation may also be made use of, by which means increased vesicular murmur will be heard in the very early stages, then later pleural friction, with dulness or absence of the respiratory sounds in the hepatized lung areas and in the parts below the mean level of any pleural exudate thrown out. Along with the increase of pleural fluid and of hepatized areas of lung there is exaggeration of the normal vesicular murmur in the unaffected parts of the lung, and the frequency of the respirations is increased up to 40 or 50 per minute. The pulse is fast and thready, between 80 and 100 per minute. The animal remains standing with neck outstretched and head lowered, coughing frequently and sometimes voiding a mucous nasal discharge. Thus there are all the signs of severe pneumonia with pleurisy and effusion of fluid into the chest. In the later stages there may be œdema under the sternum and along the floor of the abdomen.

In such an attack of what may be called the acute type the animal usually dies from asphyxia and exhaustion in from ten to twenty days after the commencement of symptoms.

On the other hand, in quite a considerable percentage of these acute cases the animals appear to recover. The symptoms gradually abate, appetite returns, and the animals put on condition, and yet when slaughtered months afterwards may be found with large encapsulated necrotic areas in the lungs. Such animals are potential centres of infection throughout this time.

In other cases the acute stage gradually merges into a chronic one in which, although the temperature returns to normal, the breathing remains somewhat hurried, especially on the slightest exertion. Coughing is easily induced, and solid areas can still be detected in the lungs by means of percussion and auscultation. In these cases also necrotic patches will generally be found in the lungs. The animal ceases to thrive, gradually becomes emaciated and weak, and often dies in two or three months' time. During this time such an animal is usually highly dangerous in spreading infection.

Besides these typical acute cases, one may expect in any large outbreak to meet with a small number of hyperacute cases, in which the onset is much more acute, the febrile period earlier, and where death may occur in a week to ten days from the time of onset. In such cases there is usually well-developed pneumonia affecting a large area of both lungs with abundant outpouring of pleural exudate.

On the other hand, a few of the cases developing in the usual way appear to be of a very mild type. There are no acute symptoms, and after an illness shown by a slight rise of temperature lasting a few days the fever abates and the animal returns to normal health. These animals seem to be completely recovered, and if kept under observation they are often noticed to put on condition rapidly and fatten.

In any but these abortive cases complete recoveries are very rare, and indeed with definite hepatization of lung tissue it is unlikely, as stated elsewhere, that resolution ever takes place.

The progress of the epidemic through a herd is slow, the cases occurring two or three at a time in a series of crops. With a large number of cattle on a holding or station the disease may take several months to make its way through the herd unless some steps are taken with a view to eradication or prevention. The importation of fresh susceptible stock at intervals will tend to keep up the contagion and prolong the outbreak.

Lesions.—On opening the chest of an animal dead of pleuro-pneumonia or killed at the height of an acute attack the first abnormality to be noticed in the majority of cases is a sero-fibrinous pleurisy with effusion of fluid. The pleural cavity contains a variable amount of exudate, up to 2 or 3 gallons sometimes, amber coloured, and with shreds or flocculi of fibrinous lymph floating in it.

In some few cases there is no pleurisy ; in others the pleura is simply lustreless, hyperæmic as if injected, and covered with a fine lace-like fibrinous layer. Usually, however, the visceral pleura is much thickened and covered with a layer of creamy fibrinous lymph which is inelastic and friable. On section the thickened pleura is found to be infiltrated with serous exudate. The parietal pleura is often only little altered.

The pericardium may be similarly involved.

The chief lesion is, however, in the lungs. There is usually extensive solidification of one or both lungs, and the affected part is much swollen and stands out above the level of the non-affected portion. On cutting into the hepatized part a clear serous straw-coloured exudate—the pleuro-pneumonia " lymph "—escapes in large quantity. It may be tinged with blood and it usually coagulates on standing. The cut surface of the lung presents a very characteristic " marbled " appearance due to the great thickness of the interlobular connective-tissue septa. These septa, thin and delicate in the normal lung, may be up to 2 or even 3 centimetres thick. They are apparently firm, but on being cut across are found to be infiltrated with the same clear straw-coloured exudate, and this distends the lymph spaces and vessels of the septum. The thickened septa surround polygonal spaces or islands of lung parenchyma and these are very characteristically of varying colours, from rose pink through yellow and orange, bright red and dark red, to nearly black. The lung tissue owes its altered structure and appearance in part to the inflammatory process affecting it and in part to the compression to which it is subjected by reason of the infiltrating and abundant exudate. The pulmonary veins running through the area are plugged with thrombus (blood-clot). Towards the periphery of the lesion the septa are less thickened but the changes in the septa extend further than do those in the lung tissue proper, so that apparently normal lung tissue may be seen outlined by thickened and infiltrated strands of connective tissue. A study of the lesions shows clearly that the inflammatory process affects primarily the interlobular connective tissue and that the lobules are attacked secondarily. The bronchioles in the hepatized areas appear to be somewhat dilated, and their connective tissue covering is thickened and infiltrated with serous exudate. The bronchial and mediastinal glands are much swollen, and are soft, succulent, and œdematous on section.

In the more chronic cases the pleura is almost invariably involved. There are numerous old adhesions between visceral and parietal layers, and there is less free fluid in the chest. On section the pleura is very much thickened and firm from the formation of inflammatory fibrous tissue in its depth. Similarly, the interlobular septa in the lung, in which the thickening was in the first instance due to serous infiltration, are now stiffer by reason of the formation of inflammatory fibrous tissue. The lung parenchyma is, generally speaking, of a paler (dirty yellow or buff) tint in these more chronic cases than in those of the acute type, and there is much less free fluid poured out on section.

A further change which occurs in a considerable proportion of all but the very acute cases is that of necrosis of lung tissue. This may involve a larger or smaller portion of the affected lung, in some cases a very large part. The necrosis is probably due to a combination of two causes, namely, the direct toxic action of the causal organism, and the thrombosis of the vessels of the part. The dying tissue area becomes somewhat dryer and tougher than before, paler in colour, and is soon definitely marked off from the surrounding living tissue by a zone of reaction. There is an abundant invasion by leucocytes into this zone, so that the " sequestrum " of dead tissue is after a time detached or separated from the living tissue. A fibrous capsule is then developed further out in the attempt to shut off the sequestrum completely. If this dead tissue, containing the virus of pleuro-pneumonia in a still active form, were completely and finally isolated by means of the fibrous tissue capsule, then with recovery of the animal the presence of such an encysted piece of dead lung might be, perhaps, of little or no consequence. Unfortunately the cavity may and does, in some cases, communicate with a patent bronchiole. In other cases the insulation is only temporary, and weeks or months afterwards with the onset of some other disease of the respiratory organs, it may be tuberculosis or a simple bronchitis or sporadic pneumonia, or even as the result of coughing, the "cyst" wall gives way at some part and either the disease (pleuro-pneumonia) may be relighted up in the same animal, or this apparently recovered animal may become infective to other healthy but susceptible cattle. It is somewhat remarkable that lesions of two and in rare cases of three distinctly different ages may be seen in the one lung, pointing to one or two re-infections. It is because of the frequency of these encysted necrotic areas in the lungs, and because of their undoubted infective rôle in many outbreaks of the disease, that recovered cattle should always be looked upon as potentially dangerous.

Microscopic Characters of the Lesions.—The primary change must be considered an infiltration of the lung connective tissue with serous exudate. Thus a characteristic feature is the dilatation of the lymph spaces and vessels in the connective-tissue septa which are filled with fibrinous lymph. This is seen under the microscope in the form of fine threads which entangle a few leucocytes. In older lesions the threads of fibrin may be seen broken down into granules; there may be many leucocytes in the part for the purpose of clearing up the dead tissue; and later still fibroblasts will be recognized laying down the new fibrous tissue.

The changes within the islands of lung tissue are evidently secondary to those in the connective tissue. The type of pneumonia is distinctly of the croupous variety and a fibrinous exudate, here again appearing as a network of fine threads, fills the air vessels adjacent to the septa. The central vesicles in a lobule may be almost normal, or possibly somewhat compressed. Some of the vesicles may show a certain amount of catarrhal change with proliferation and shedding of epithelium, especially in the more chronic lesions. Some areas have the vesicles filled with blood corpuscles, and such correspond with the darkest islands of lung tissue in the lesion.

The bronchi and bronchioles may show some evidence of catarrh with proliferation and shedding of epithelium, dilatation of capillaries, and in more chronic cases some thickening of their walls with fibrous tissue.

Lesions produced by Experimental Infection.—With pure culture or virus injected subcutaneously the lesions which develop show a characteristic likeness to those in the lung in natural cases. The reaction occurs not only in the subcutaneous areolar tissue, but extends to the underlying muscles. The intermuscular fascia is thickened and infiltrated so as to produce in the muscle an appearance of "marbling," whilst the muscle fibres themselves have a parboiled appearance. There is an abundant exudate, subcutaneous, intermuscular and interstitial.

In young calves infected naturally it is not unusual to see a multiple arthritis, of a sero-fibrinous variety.

Diagnosis.—In an isolated case or in the earliest case of an outbreak a positive diagnosis of contagious pleuro-pneumonia is not easy to make. The signs and symptoms are simply those of pneumonia and pleurisy with somewhat insidious onset. If the contagious disease is known to exist in the country or district it will probably be thought of, but until a post-mortem examination is possible or infection of other cattle occurs a positive pronouncement cannot be made. If other cattle have been in contact with the sick animal up to the onset of illness these animals will be examined (1) by means of temperature records and (2) by being driven or chased in a field or yard to determine any undue disturbance of respiration, and the occurrence of the characteristic cough on coming to rest. An elevation of temperature of 2° or 3° F. in an animal apparently healthy is very suggestive, and if exertion induces coughing and rapid breathing out of proportion to the exercise, the suspicion becomes almost a certainty.

Several other diseases may give rise to symptoms similar to those of pleuro-pneumonia and require to be differentiated from it. Of these there are:

1. **Tuberculosis.**—In this case the disease comes on more gradually, there is little elevation of temperature, the cough is chronic and of a moist character, but there is an absence of pain on percussion of the chest, and no evidence of the outpouring of fluid into the pleural cavity as a rule. The occasional acute cases of pulmonary tuberculosis on the one hand, and the chronic types of pleuro-pneumonia on the other hand, present the greatest difficulty. A negative tuberculin test eliminates the possibility of tuberculosis, and absence of contagion to other cattle in contact practically negatives the diagnosis of pleuro-pneumonia. It must be remembered that the two diseases frequently occur together in the same animal.

2. **Traumatic Pericarditis.**—This may occasionally present some difficulty, for the attitude is similar, and the typical grunt on rising, together with the quickened respiration, suggest pleuro-pneumonia. On the other hand, the early history of the case with symptoms of recurrent colicky indigestion; the more localized area of pain on percussion limited more or less to the pericardial area; the quickened pulse and muffled heart-sounds; friction synchronous with the heart-beats; and the œdema of the dewlap extending up the jugular furrows, determine the diagnosis of pericarditis, whilst the absence of contagion is against pleuro-pneumonia.

3. **Sporadic Pleurisy and Pneumonia.**—This is as a rule more acute in its onset, the case is an isolated one, and its course is more rapid to recovery or death. On post-mortem the lungs present a different picture in that the interlobular septa are usually much less thickened, infiltration of the septa with fluid is not a feature, and the lung parenchyma in the affected areas does not show the variation of colour so characteristic of contagious pleuro-pneumonia.

4. **Hæmorrhagic Septicæmia.**—The pectoral form of this disease presents some similarity to pleuro-pneumonia in its lesions, but not much likeness as a rule during life. In the ordinary case the septicæmic disease is much more acute, coming on rapidly and running its course often to a fatal issue in from five to eight days. The symptoms and signs are not confined to the lungs and pleura but point to bowel lesions as well. In some subacute to chronic cases, however, there may be difficulty, and on post-mortem the lungs may bear a distinct resemblance to those seen in pleuro-pneumonia. Distinguishing features are an absence as a rule of any marked variation in the colour of the islands of lung tissue in the hepatized part; and the presence in the lung and pleural exudate of bipolar staining bacilli (*pasteurellæ*), which on inoculation are usually virulent for rabbits and calves. Pleuro-pneumonia lymph, on the other hand, is innocuous for rabbits, and in calves it develops the characteristic lesions as already described. Exudate in septicæmia which fails to show any organisms on microscopic examination will often do so if taken up in a sterile pipette, sealed, and incubated for twenty-four hours.

Immunization—Preventive Treatment

No curative treatment is permissible in cases of pleuro-pneumonia. Animals affected with the disease must be slaughtered as soon as the diagnosis is confirmed in order to confine the possibility of infection within the narrowest possible limits. Other healthy cattle may be treated by one of several methods with a view to rendering them insusceptible.

1. *Willem's Method.*—This, the oldest method, is still largely practised in countries where pleuro-pneumonia exists. An animal just dead of the disease or one killed at the height of an acute attack is taken, the chest opened and the affected lung exposed. The lung should show the characteristic hepatization and infiltration, but should not contain necrotic areas. On incising the hepatized part deeply the clear amber-coloured lymph exudes and collects in the incision. It is collected in sterile tubes or suitable vessels, and forms the inoculation material, without further preparation if to be used immediately. The site of inoculation is near the tip of the tail, the hair is removed for a space of two or three inches, the skin disinfected with iodine solution, and a strong grooved needle, freshly dipped in the "lymph," is inserted subcutaneously for a length of about one inch. This is a very simple but quite effective method of inoculation. Various hypodermic syringes specially adapted for this purpose have been devised, and are so constructed as to inject two or three drops of lymph. The percentage of reactions obtained with these instruments is not, however, appreciably greater than with the simple needle.

When not to be used immediately, steps must be taken to preserve the "lymph," and this is done by the addition of one volume of glycerine and one volume of 0·5 per cent phenol to two volumes of fresh lymph. Such glycerinized lymph remains active for two to three months (Nocard).

The reaction is seen in from six to twenty days, and when, after eight to ten days, this has subsided the animal is immune in that it will not respond to experimental infection with either lung lymph or pure culture.

2. *Pasteur's Method.*—Because of the difficulty of obtaining lung lymph in the field in a state of purity, uncontaminated with accidental organisms, or with tubercle bacilli in cases of dual infection, Pasteur devised a method by which large supplies of lymph could be obtained with aseptic precautions. A calf four to six months old is inoculated with a few cubic centimetres of virulent lymph in some region where there is plenty of loose subcutaneous tissue, commonly in the skin over the chest just behind the elbow. A large swelling is produced following a febrile reaction, and when the swelling has attained its maximum the calf is destroyed, the skin shaved and cleansed, opened with aseptic precautions, and the infiltrated connective tissue incised. Lymph containing the active virus collects in the incision and is at once drawn up into sterile pipettes which are sealed up. This calf lymph may then be used for tail inoculations just as in Willem's method. It has the advantage of purity, but provokes a milder reaction and probably a lesser immunity than the natural lymph. Such lymph will remain active for four to six weeks if kept in a cool place.

3. *Nocard's Method.*—After the successful cultivation of the pleuro-pneumonia organism was achieved the difficulty of supplying fresh lymph, whether natural or from a calf inoculated for the purpose, was no longer of moment, for Nocard suggested the use of pure cultures of the organism as vaccine. Broth cultures are used in doses of from 0·25 to 0·5 c.c. injected near the

tip of the tail with a hypodermic syringe, and in France, where a large number of inoculations with pure culture have been performed, the method has proved satisfactory if not superior to the more crude methods used formerly.

4. *Ingestion Method.*—A method which has been practised by the Boers in South Africa for many years is that of drenching healthy cattle with 5 or 6 ounces of virulent lung lymph. This method has the practical disadvantage that the supply of suitable lymph must be large, and further, it is much more troublesome than the tail injection method. Moreover, Nocard's experiments suggest that this method is useless.

5. *Hyper-immune Serum Method.*—When cattle have been immunized by the tail inoculation method, they will withstand injections of lymph or pure culture. If such injections are gradually increased and repeated at weekly intervals for some months the serum of these animals becomes hyper-immune and will confer a passive immunity lasting one to two weeks on healthy cattle, in that they will not react to subcutaneous injection of pure cultures. Further, if a subcutaneous injection of virulent material has already been made, the use of hyper-immune serum will render it abortive. The dose, however, requires to be large, 40 to 50 c.c. or more; the preparation is expensive, and the immunity so short-lived that the method is of little or no practical value.

Veterinary Police Measures.—Contagious bovine pleuro-pneumonia is of so serious a nature that it is subjected to rigorous legislative and administrative regulations in whatever countries it exists. There are in the main two lines of procedure which may be adopted, namely (*a*) the method of compulsory slaughter of all affected animals together with all "contacts," and those suspected of being infected; and (*b*) the compulsory slaughter of those affected only, with immunization by means of inoculation of all animals which have been in contact with the affected animals or are in the declared "infected area."

The first method has undoubtedly justified itself in Great Britain, Germany, Austria-Hungary, and other countries, where it has resulted in complete eradication within a few years, in several cases after the inoculation method had been tried for some years and had failed to eradicate the disease. The method is costly, and in a country dependent on the dairying industry and the meat trade it is attended with great difficulties, since it may entail considerable depletion of herds in some cases.

The second method is less costly of application, at any rate at first, but it is doubtful whether it is so in the long run. The process of eradication is less certain, and a much more lengthy one by this method, but that it may succeed is evidenced by the case of France, where it was adopted.

In Australia a modified form of this method is applied with a considerable degree of success and at a minimum cost. Affected animals are slaughtered, in-contacts and cattle on neighbouring holdings are inoculated with a view to immunization, and all in-contacts are quarantined on the farm, only being removed to an abattoir for slaughter under licence

H. A. W.

EPHEMERAL FEVER

Three-day Sickness; Stiff Sickness; Dengue

Definition.—An acute febrile disease of cattle, usually running its course in three days and characterized by muscular stiffness.

History.—This disease was first observed among cattle in North-Western Rhodesia, and made its appearance in Matabeleland during the latter part of November 1906, from whence it rapidly spread to Mashonaland, where it was investigated and first described by Bevan in the early months of the year 1907. From Rhodesia it gradually spread southward until it reached the Transvaal and Natal, and since then the disease has reappeared from time to time, its occurrence apparently being associated with, and dependent upon, the severity of the rainy season. The disease also occurs in Egypt and has been described by Piot-Bey, and a similar malady is also said to be common in India during the rains.

The incidence of the disease is remarkable; outbreaks occur in areas far removed from one another, and between which there has been no interchange of stock. Cattle of all ages, of both sexes, in high and low condition, are equally susceptible; trek oxen, cattle at pasture and milch cows suffer alike. No particular breed of cattle appears to possess any immunity; the native cattle of Mashonaland and Matabeleland, as well as imported animals from the Southern territories and from the North, and recently imported animals from Great Britain, are subject to the disease. Cattle salted to "Red-water," "Anaplasmosis" and "East Coast Fever" possess no immunity. Animals known to have contracted the disease in 1907 have suffered again in subsequent outbreaks. Fat animals and bulls appear to suffer more severely than those in low condition, but the disease is by no means peculiar to fat animals.

The disease makes its appearance suddenly in a herd, and it is then noticed that within the first day or two quite a number of the animals become affected; it appears to originate spontaneously. For this reason it has been suggested that a peculiar condition of the pasture or of the atmosphere may be responsible, but although these may play a part in the occurrence of the disease, they are by no means the only factors. It has been noticed in the Cape Colony that stabled animals suffer from the malady, and a case was noticed in a bull which was closely housed and fed on certain dry foods which were brought to it, with a view to testing their feeding values.

The specific cause of the disease is present in the blood of the sick animal, and during the 1907 outbreak Bevan sent a quantity of preserved blood to Robertson, of the Grahamstown Laboratory, who inoculated the same into the jugular vein of an ox which developed the typical temperature and characteristic stiffness of the limbs. This experiment was repeated by several observers with similar results, and the *period of incubation* is now known to be from two to three days. The attack so conferred is followed by an immunity lasting for at least six weeks, but the blood of a recovered animal does not produce the disease if taken when all the symptoms have subsided. Nevertheless, the incidence of the disease would give rise to the suspicion that the specific organism is present in bovines, producing no ill effects in them until some unusual conditions reduce the tolerance or immunity of the animal. An analogy to this is met with in the well-known tolerance of fly-struck cattle to trypanosomes, until exposure to heavy rains breaks down their immunity, often with fatal effects. It has been suggested that the transmission of the disease in nature is through the agency of night midges, and such an explanation would account for those cases which occur in stables and in towns.

Symptoms.—As the popular name "Three-day Sickness" suggests, typical cases of the disease run their course in about three days, but in complicated cases the symptoms may persist for weeks. On the other hand, acute cases have been known to recover in twenty-four hours. At first the disease was given the name of "Stiff Sickness" because the prevailing symptom encountered was stiffness. The confusion, however, between the three-day sickness and the stiff sickness or "Lamziekte" of cattle, has rendered it desirable to avoid the term.

The stiffness may be noticed in one or more legs. It may involve one or both fore legs or one or both hind legs. A fore limb and a hind limb may be simultaneously affected, or all four legs may be stiff at the same time. The stiffness may rapidly pass from one limb to another, and it has been observed that a trek animal will pull up lame in one limb and move off lame in another. The stiffness may involve the muscles of the neck or back, and when the former are involved the animal stands in a very characteristic attitude, with its head protruded and sometimes with its lips drawn to one side. When the limbs are involved the animal shows a disinclination to move, and it does so only with great discomfort, and with the same amount of difficulty and with the arching of the back seen in horses suffering from laminitis.

When the animal is at rest it is often possible to detect the affected limb by the appearance of rigidity or flatness of the muscle bellies. They may feel hard or tense to the touch, and there is often a sort of quivering or pulsation in or around them. In one reported case it appears that the diaphragm was involved, as the animal was said to be suffering from "hiccup." In many cases there is a certain amount of swelling around the large joints of the affected limb, but this is not a constant feature. It has also been remarked that the part of the animal affected can be determined by the manner in which the flies settle on it; and a hardness and swelling of the skin over the affected area occurs to such an extent that it is often impossible to pinch the skin into a fold, or having done so, the fold remains for a considerable time.

The stiffness of the muscles of the neck occasionally extends to those of the pharynx, so that deglutition is rendered difficult or impossible. In one case seen the tissues between the lower maxillary bones were swollen and œdematous, recalling the condition often seen in "dikkop" cases of horse-sickness. In one case the animal was lame on the first day in the near fore leg, and on the second day this had passed away and a swelling appeared on the side of the neck; later, during the second day, vertigo set in, and the animal constantly turned in a circle to the left until it fell exhausted. It is popularly believed that the lameness does not become acute if the animal can be kept moving; this is not invariably the case.

The coat of the sick beast is not always harsh or dry, and the muzzle is generally quite moist and covered with a healthy "dew." There may be some ropy saliva hanging from the lips, and the inner lining of the mouth is usually somewhat red.

The animal frequently grinds its teeth and groans, and when lying down allows its head to fall to its side in the position seen in cases of milk fever. During the first day of sickness the beast neither feeds nor ruminates; the return of appetite and chewing of the cud may be taken as a sign of recovery; this is generally observed on the second day.

A very constant and noticeable feature is a swelling around the eyes, which gives the animal a dull, heavy appearance; in many cases this swelling and severe lachrymation is the earliest clinical symptom, and with redness of the conjunctiva and glaring appearance of the eye is most diagnostic. In some cases heavy respiration and a copious flow of fluid from the nostril is noticed, especially at the onset of the trouble, but difficult breathing is not a constant symptom. In animals lying down respirations are shallow and interrupted by an occasional deep breath. A jugular pulse is very often well marked, and the heart-beats are weak and difficult to register. At the onset of the disease the temperature is greatly elevated, and may reach as high as 106·5° to 107° F. In cases running a normal course it rapidly declines, and on the second day it is seldom above 104° F. Constipation is not an invariable feature; frequently it happens that the animal does not defæcate for a day or two and undue importance has been given to this fact. Indeed, diarrhœa is not uncommonly present, and in some cases dysentery has been observed. In the majority of cases the dung is dry and lumpy and covered with great strings of mucus. Tympanites is seldom present but has been recorded.

In typical cases the disease runs its course in three or four days, a circumstance which has given it the title of "three-day sickness." In complicated cases it may persist, and animals have remained ill for three weeks and longer. When once the appetite returns and fæces of normal consistence are voided recovery is regarded as assured.

Post-mortem Appearances.—Owing to the extremely low mortality and to the fact that the deaths have chiefly occurred among those cases where the muscles of the neck have been involved and medicines have passed down the trachea, or where drugs have been given causing various complications, it is difficult to describe the lesions which are proper to the disease. In most cases a copious blood-stained discharge has been found escaping from the nose, and a quantity of froth and saliva accumulates around the lips. On removing the skin the inner surface has been stained with blood, and it has sometimes been observed that this discoloration has been more marked in the region where the animal has suffered from stiffness during life. The blood is found clotted firmly in the heart and vessels, and will protrude stiffly from the vessels when they are cut, or will escape in long black cords. In some cases, on cutting the lung tissue, these clots have projected in a very remarkable manner, sticking up from the cut surface like slate pencils. The arterioles throughout the body are congested and their ramifications can be easily made out. Indeed, the alterations observed in the tissues appear more or less dependent upon this state of the blood, and to the fact that the serum expressed from the clots is coloured with the red colouring matter. The muscles of the carcase have appeared cloudy or muddy in those places not blood-stained, but in other parts have been of brick-red colour, due to a quantity of blood-stained fluid which has saturated them. In those cases where during life the muscles of the neck have been involved a swollen condition of the inter-maxillary tissues has been well marked, and on section these have been found blood-stained and emphysematous. The lungs have been found congested and undergoing putrefaction. This may have been due to medicines which have passed down the "wrong way." With one exception the heart sac contained a quantity of blood-stained fluid, the epicardium showed hæmorrhages, and the myocardium was deeply stained by the blood-coloured fluid previously mentioned. The blood in the heart cavities and large vessels was very black and had clotted, but the expressed serum was dark red in colour. The liver, kidneys, bladder, and spleen showed no lesions. The omentum in all cases was discoloured; the fat was reddish-yellow and the membrane was brick-red wherever the blood-vessels were most numerous. The rumen, reticulum, and omasum appeared normal and contained a quantity of ingesta of normal consistence. The contents of the omasum were not dry or impacted. In the fourth stomach (abomasum) the principal lesion was constantly met with in the form of an intense magenta discoloration of the mucous membrane, recalling the condition of the stomach of animals dead from horse-sickness. The gall-bladder was full of gall of normal appearance and consistence. The urine was light-coloured and there was no evidence of hæmoglobinuria. The brain appeared slightly congested and was stained pink by the effusion from the blood-vessels. An abnormal quantity of reddish cerebro-spinal fluid was present in one case.

To summarize, the chief pathological changes encountered have been: (1) an altered condition of the blood, which clots very readily and which allows a ready effusion of red-coloured material into the surrounding tissues; (2) a quantity of blood-stained fluid in the pericardial sac; (3) a violent inflammation of the lining of the abomasum.

Mortality.—The great majority of cases recover, and it is probable that the mortality would be even lower if it were not for complications commonly caused by injudicious treatment. The dosing of animals unable to swallow is frequently followed by pneumonia and other fatal results. Nevertheless, it has been observed that during an outbreak the disease will become exalted in virulence, and in certain herds of improved breeding it has been known to assume a very fatal form, animals having been found dead a few hours after the first indication of the disease.

Cause of the Disease.—This has not yet been determined, but the infective agent exists in the blood of an actively infected animal and can be transmitted from sick to healthy by inoculation.

Quantities of citrated blood were injected by Bevan into a donkey, a rabbit, a chicken and sheep. Only the last of these yielded a reaction. In the latter a well-defined thermal elevation was apparent between the sixth and tenth days. This reaction, however, was not accompanied by stiffness or lameness, and sheep which had reacted to such an inoculation failed to react to a second injection of virulent blood.

A similar temperature reaction was also obtained in sheep inoculated subcutaneously with an emulsion of spinal cord taken from an ox which had died of so-called stiff-sickness.

As previously mentioned, blood from a sick animal in Salisbury was sent to Robertson, and on arrival at Grahamstown proved capable of setting up the typical disease in cattle inoculated by him.

Immunity.—Animals after recovery possess some degree of immunity, but this is said to be of a transient nature. It was generally believed that animals which had suffered from the disease during one outbreak were immune, at any rate for that season, and it was rare to find an animal suffering from a second attack of the malady. Freer, however, states that he has seen affected cows, the owner of which assured him that they were suffering for the fourth time. In his opinion, in the majority of cases, the second attack is worse than the first.

Treatment.—No specific remedy has been found and the great majority of cases recover without treatment. Indeed, it is advisable in most cases to avoid administering medicines for fear of the complications which may be set up thereby. The sick animal should be protected from extremes of heat, cold and rain, and if constipation is present enemata may be administered with advantage. Beneficial results have also been known to follow the sweating of the animal by means of blankets and rugs.

LL. E. W. B.

FOOT-AND-MOUTH DISEASE [1]

Eczema Epizoötica ; Epizootic Aphtha ; Aphthous Fever

Foot-and-mouth disease is an acute contagious fever affecting cattle, sheep, goats and swine, and associated with vesicular lesions about the mouth or feet, or both. Horses, poultry and human beings are also susceptible, but to a very much less degree. As in the case of other contagious diseases, foot-and-mouth disease never arises spontaneously, and outbreaks are always attributable to some previous case of the disease. It is caused by an ultra-microscopic filtrable virus which has up to the present not been artificially cultivated. It is present in the lesions, and is liberated when these burst. In this way saliva, hides, hair and manure may all become contaminated. The method of spread of foot-and-mouth disease appears to be variable, and it is generally agreed that it may be conveyed by hay, straw, hides and skins, carcases of calves in skins, milk and milk products, drovers and their clothing. It may also be conveyed in the form of vaccine lymph and in this way it may be maliciously propagated. It has also been suggested that birds may actually convey infective material on their feet after contaminating them in an affected place. The natural method of infection is by the alimentary tract. It is probable that it may also be contracted by inhalation, while experimentally it can be transmitted by intravenous inoculation.

The period of incubation is from one to ten days, the most common period being between three and six days.

Symptoms.—The first indication is general indisposition on the part of the animal with loss of appetite, and in the case of milch cattle a reduction in the milk yield. The temperature rises rapidly to 104°-106° F., but the high fever only lasts a day or two until the local lesions develop, when it subsides. Vesicles next begin to form about the mouth and round the hoofs. This gives rise to dribbling of stringy saliva and smacking of the lips ; indisposition to feed, which appears to be mainly due to the pain and not to the lack of appetite, since animals given soft food will frequently eat. There is rapid loss of condition. The development of the foot lesions causes pain and lameness with shaking of the feet and kicking.

The specific lesions are the same in each of the different classes of animals affected. They consist of the development of vesicles or blisters, the epidermis or the epithelium of the mucous membrane being raised from the underlying structures by a clear watery liquid. In cattle, as a rule, the mouth and feet both show lesions, while in sheep and swine the foot lesions are much more common than lesions in the mouth, though both may occur. The size of the vesicles varies : at first small about the size of a split pea, they may rapidly increase until they have a diameter of a five-shilling-piece, but average about one inch. In the mouth the lesions are mainly seen on the tongue, but may also be met with on the lips and dental pad, and on the snout in swine. The mouth lesions are usually circular unless several vesicles have coalesced, in which case their outline is irregular. They are white on the surface when first seen and intact, but when they burst, a red, smooth surface is exposed with ragged edges. In this way almost the entire free portion of the tongue may become denuded of its epithelium. Sometimes the muzzle shows an accumulation of epithelium and may be rubbed raw. When the lesions heal, no permanent marking is left, unless other complications such as suppuration have occurred.

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[1] The illustrations for this article are reproduced from the Report of the D.A.T.I. on Foot-and-mouth Disease in Ireland in 1912.

When the feet are affected in cattle, the lesions usually commence at the top of the hoof in the cleft of the foot, while in sheep and swine they more frequently commence at the heel or at the toe. The shape of the feet lesions is elliptical. When they burst, a red sore is exposed. In some cases the exudate passes between the hoof and sensitive structures, and may result in the total loss of a claw. The foot lesions cause great pain, and affected animals can hardly walk. Cattle frequently lie down and refuse to rise, while sheep and swine sometimes move about on their knees. In milch cattle lesions may also be met with on the udder and teats, and sometimes the point of the teat may be surrounded by a single vesicle. The characters of the vesicles in this situation are the same as elsewhere; first white blisters which soon burst, especially in milking, exposing a red sore. The teats themselves are usually swollen and painful, and the secretion of milk is much diminished.

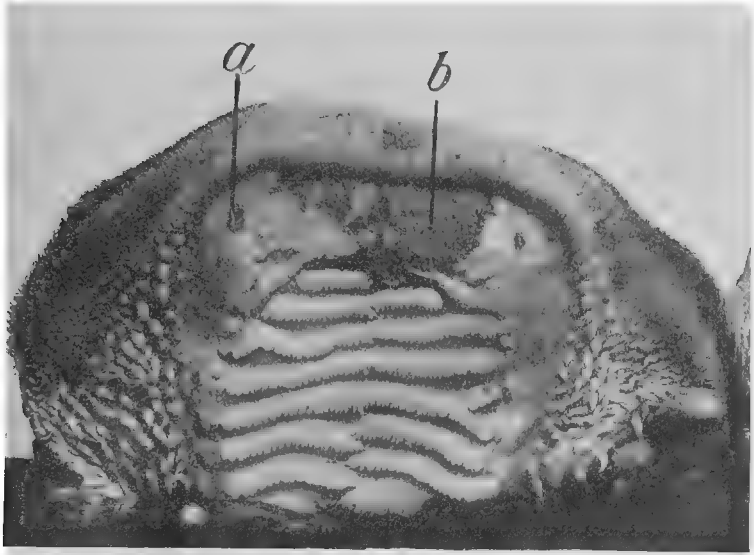

Fig. 23.—Dental pad and portion of palate of cow with foot-and-mouth disease. *a*, Small ruptured vesicle. *b*, Erosion of mucous membrane, following rupture of vesicle.

In very young animals the disease is much more likely to prove fatal than in adults, and death may occur before the specific lesions have had time to develop in either the mouth or the feet. In such cases all that can be found on post-mortem examination is a superficial gastro-enteritis.

Course.—The disease generally runs through its course in from fifteen to eighteen days, though individual lesions may heal within a week. The lengthened period is due to successive lesions occurring in different parts of the body. In a herd the outbreak generally runs its course in from five to six weeks. The mortality of foot-and-mouth disease is very low, probably less than 1 per cent in adults. In young animals the death-rate is much higher, particularly if they are sucking affected parents. In rare outbreaks, however, high mortality has been reached, but that has generally been where the herd has contained a large proportion of young animals. In some outbreaks of a very malignant character, according to Bang, there may be "mortality of from 5 up to 50 per cent among adult animals, and from 50 up to 80 per cent among young animals." Recovery leaves an immunity which lasts a year or more.

Although in outbreaks the general mortality is low, foot-and-mouth disease is one of the most serious affections of the domesticated animals. The great loss is due first to the loss of milk yield in a dairy herd, and secondly to the greatly reduced condition of the affected animals of all species. Even after recovery the secretion of milk rarely comes up to the previous standard. In some cases mammitis may occur, rendering the cow useless for dairy purposes, while in other cases persistent lameness results and animals remain unthrifty. In addition one must include the heavy expenses entailed in properly looking after sick animals, and the necessity for sterilizing all the products. Another most important source of loss is the interference with trading in live stock.

Differential Diagnosis.—In an established outbreak there is little or no difficulty in definitely diagnosing foot-and-mouth disease. The rapidly contagious character of the disease, together with the fact that both classes of ruminants and swine are simultaneously affected, assists diagnosis. Difficulty, however, may be encountered with the first suspected case of an outbreak.

Lesions of the mouth may be confused with various forms of stomatitis in cattle. In Ireland one particular form which has been called **"thrush"** or "dirty mouth" has caused confusion. This form of stomatitis has been well described by Mettam as follows :

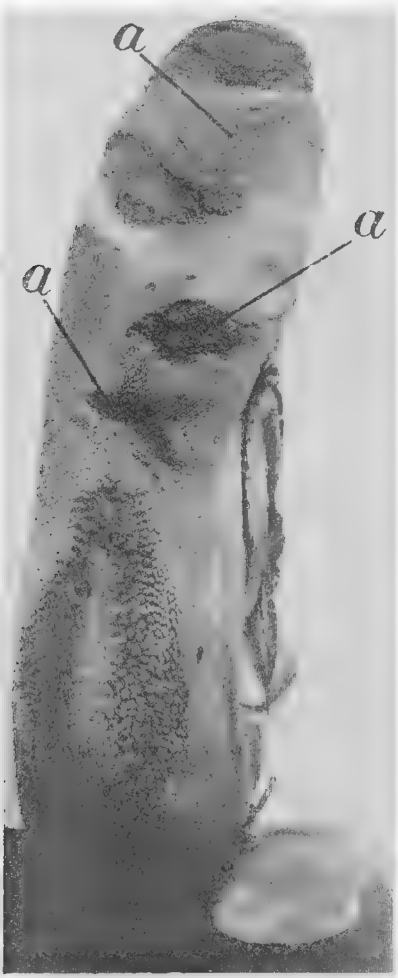

Fig. 24. — Tongue of bullock with foot-and-mouth disease. *a, a, a,* Erosions of mucous membrane after rupture of vesicles.

"This stomatitis has no effect whatsoever upon the health of the animal. It apparently does not suffer any inconvenience at all. The animal feeds quite normally. The condition is noticed at the time of 'mouthing' the animal, and until the mouth is opened and the condition of the mucous membrane attentively examined nothing is suspected. Sometimes the appearances then presented are alarming, especially when one is 'keyed up' and on the outlook for foot-and-mouth disease. The lesion begins in the superficial epithelium of the lips, gums or tongue. It at first is a brownish spot which enlarges peripherally. The epithelium involved separates off in flakes which are perforated (on the tongue) with apertures through which the papillæ passed. A number of lesions may begin simultaneously and extending, eventually coalesce. They all show the brownish discoloration and exfoliation of the epithelium. Eventually the tongue shows large areas denuded of superficial epithelium, with broad, low ridges of old epithelium persisting. These are the boundaries of adjacent lesions. In time all the old horny epithelium is lost, and the tongue becomes quite clean and soft, like that of a young calf. There never at any time is any exposure of corium, never at any time formation of vesicles, never at any time soreness of the tongue, drivelling of saliva, or other sign of irritation. There never is any lameness or other lesion upon the body even remotely suggesting foot-and-mouth infection. The condition is found chiefly in young stock, rarely in full-grown cattle, and it is not inoculable to sheep or to swine as it is to young bovines.

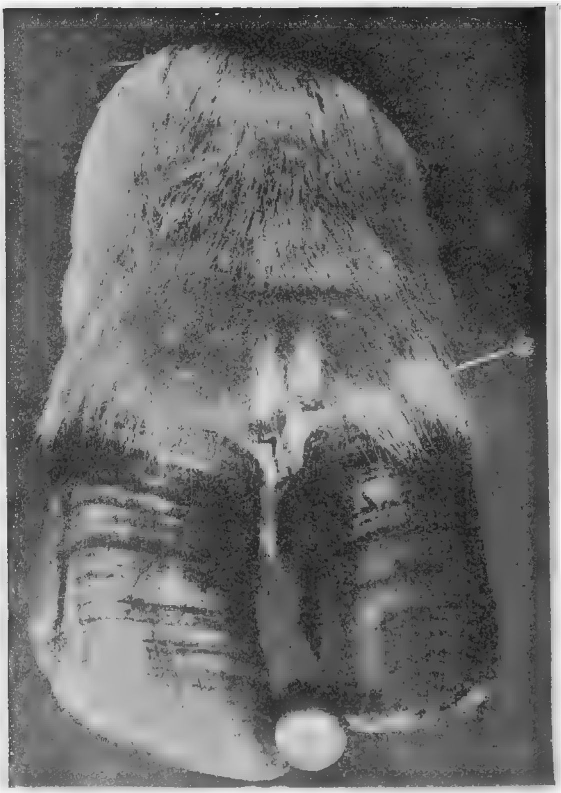

Fig. 25.—Foot of cow with foot-and-mouth disease. *a*, Ruptured vesicle at interdigital space.

The condition is not one likely to be met with by ordinary practitioners, they will never be called in to treat it. and it is only likely to be encountered by those whose duty it is to 'mouth' animals—to examine stock for mouth lesions of foot-and-mouth disease."

In sheep it may be confused with *vesicular stomatitis* due to feeding on stubble. In this condition there may be several animals simultaneously affected, but the lesions of the foot are wanting, while they are almost constant in foot-and-mouth disease.

Lesions of the foot may be confused in cattle with **foul-of-the-foot.** In this condition the lesion is necrosis or abscess formation in the cleft of the foot, and several animals may be affected in the herd. There is, however, an absence of mouth lesions and of vesicles, and there is no evidence of contagion to sheep or swine.

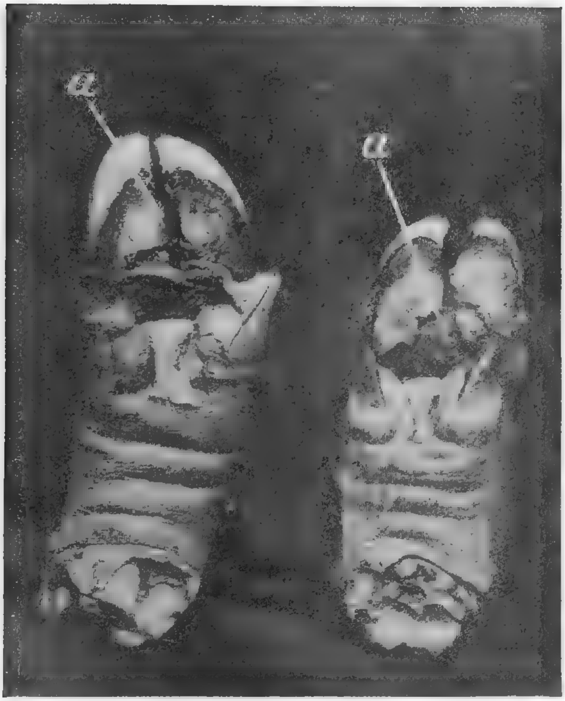

Fig. 26.—Back view of feet of swine affected with foot-and-mouth disease. *a, a,* Ruptured vesicles.

In sheep the foot lesions may be confused with **foot-rot,** the lesions of which, however, do not resemble in any way those of foot-and-mouth disease. In one form of foot-rot there may be small vesicles round the coronet, and although the condition is contagious it does not spread rapidly nor is it communicable to other species. Another form of foot-rot commences on the solar aspect of the foot, and consequently in no way resembles foot-and-mouth disease. In both forms there is absence of lesions in the mouth.

Lime-burning in sheep has been mistaken for foot-and-mouth disease. It has been caused in some cases by driving sheep through quicklime laid in a gateway in the treatment of foot-rot, and has resulted in the production of blisters round the coronets of a number of the sheep, thus simulating a contagious disease. The absence of mouth lesions and of contagion to other species, together with the history, serve to differentiate it from foot-and-mouth disease.

Treatment.—Prevention. Foot-and-mouth disease is scheduled under the "Diseases of Animals Act" in the British Isles, and all outbreaks must be reported to the local authorities and the Board of Agriculture, and no curative treatment is permitted. The procedure adopted is to slaughter all diseased animals and those that have been in contact with them, and establish a quarantine prohibiting the movement of animals over a wide area. Markets and fairs should be closed. Thorough disinfection must be practised, both in the infected farms and of all cattle conveyances which may have been used in the district by rail or road. In the surrounding districts rigid inspection should be instituted with special attention to fairs and markets. To prevent its introduction into the country, the importation of living animals is prohibited from infected countries, as also is the importation of fodder and litter. An embargo should also be placed on the introduction of hides, hair, wool, horn, manure and all commodities which may have been contaminated with infective material.

Various attempts at immunization have been made, with a view to producing *active immunity,* with a mixture of virus from active vesicles with immune serum, but the results have not been satisfactory and have even produced a serious outbreak. *Passive immunity* has been produced by the use of hyper-immune serum, but this procedure has also been abandoned. The use of hyper-immune serum may perhaps be helpful in the treatment of animals in the outer zone surrounding a district where an outbreak has occurred.

Curative Measures.—In countries where the disease is indigenous, when an outbreak occurs an attempt may be made to curtail the period of the outbreak by facilitating the spread of the disease through the flock or herd. This may be done by rubbing the lesions of affected animals with tow and then rubbing the mouths of the healthy animals with the same tow and thus inoculating them. In this way the whole of the animals are affected much more quickly than would be the case if infection was allowed to take its natural course. The inoculating material should only be taken from the milder cases.

According to the conclusions of the International Congress on Foot-and-mouth Disease at Buenos Aires 1920, the most satisfactory treatment for foot-and-mouth disease consists in inoculating with a serum prepared according to the method of Dr. Henri Marques Lisbao which has given excellent results in Brazil, where it has been employed on a large scale by the Ministry of Agriculture at Rio de Janeiro.

The lesions themselves should be treated with suitable antiseptics. For the mouth, lotions of boracic acid, alum, carbolic acid or lysol may be used, or antiseptics administered in the form of electuary; chromic acid has also been recommended. In the case of the feet, more urgent measures are required. The feet should be kept clean, the animals being put into a place where the floor is dry and with a frequent change of bedding. They should be dressed daily with disinfectant astringents such as copper sulphate or zinc chloride, or corrosive sublimate solutions. Where a number of animals are affected these may be applied by means of the foot-bath. The coronets may then be dusted with a dry powder such as tannic acid and charcoal, or oxide of zinc. Where the udder is affected the lesions may be dressed with lotions similar to those used for the mouth. In painful conditions of the teat it may be necessary to use a teat syphon for milking, in which case care must be taken to render it absolutely sterile before use, otherwise mammitis may result.

With regard to internal medication purgatives should be avoided and febrifuges may be administered, such as quinine and the salicylates. Various other remedies have been recommended, such as the intravenous injection of perchloride of mercury and also of collargol, but these methods have not been followed by favourable results, while the mercurial injections have actually caused deaths from mercurialism.

G. H. W.

SWINE FEVER

Synonyms.—Hog cholera, pig typhoid, peste du porc (*Fr.*), peste porcina (*Ital.*), Schweinepest (*Ger.*), peste de los cerdos (*Sp.*), svinpest (*Scandinavian*).

Definition.—Swine fever is a highly contagious febrile disease which affects swine only, and is caused by a filtrable virus.

History and Distribution.—The source and early history of swine fever are largely matters of conjecture, for only in the last thirty years has this disease been sufficiently well defined to permit of its differentiation, with certainty, from other affections of swine. Severe and fatal epizoötics undoubtedly prevailed among the swine in continental Europe prior to the year 1830. In the United States a similar disease was first recorded in 1833, while fatal swine diseases of the same general characteristics prevailed in England also in the early part of the last century. About the year 1880, coincidentally with the development of the science of bacteriology, the first beginnings were made in the differentiation of the several swine diseases. The researches of Loeffler first established the cause and permitted the identification of swine erysipelas, while the contemporaneous work of Salmon and Smith in the United States led them to the conclusion that there existed two other important diseases of swine which they designated respectively hog cholera and swine plague. These three diseases are recognized to-day as distinct and independent diseases of swine, though our ideas of their distribution and their relative economic importance have been greatly modified. Swine fever (the hog cholera of Salmon and Smith) has assumed vastly greater importance than was hitherto attached to it, and veterinary authorities generally have come to the opinion that the successful control of swine fever will mean the removal of the greatest menace to the hog industry.

Swine fever exists in practically all parts of the civilized world. Continental Europe, Great Britain and Ireland, South Africa, North and South America, have been infected for years, though the disease is not always equally prevalent. The very distinct periodicity of prevalence in the United States is well illustrated by the accompanying chart. The financial losses suffered by farmers from this disease amount to billions of dollars. In the United States, which is the largest swine-growing country in the world, the average losses for more than thirty years are believed to exceed £30,000 annually.

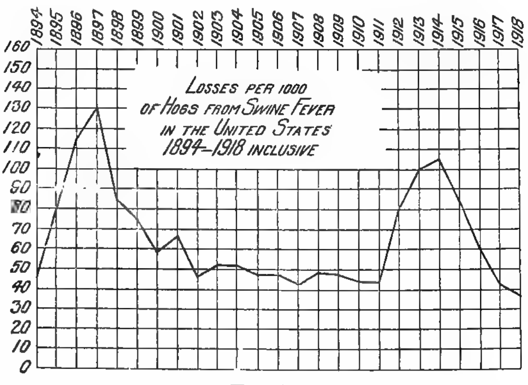

Fig. 27.

Etiology.—It is now a universally accepted fact that swine fever is caused by a filtrable virus. It is true, however, that the filtrable virus infection is usually complicated by other infections which invade the body of the pig after primary infection with the filtrable virus. The most common among such secondary or accessory factors in the disease are the swine fever bacillus (*B. cholera suis*, *B. suipestifer*) and the swine plague bacillus (*B. suisepticus*). While both of these micro-organisms are undoubtedly capable of doing much damage once they have gained a foothold in the body of the pig, neither is necessary to the development of swine fever.

The filtrable virus is found in the urine, fæces, eye and nose secretions, and in the blood. Both the blood-serum and the blood-cells contain the infection. No one has yet succeeded in growing the filtrable virus artificially, nor in staining or otherwise rendering it visible under the microscope. The virus in the blood-serum of sick hogs, particularly after dilution with bouillon or physiological salt solution, passes readily through Berkfeld and Chamberland filters of the finest type, and regularly produces disease in pigs after subcutaneous injection. No other animals are susceptible. The filtrable virus of swine fever as it exists in the blood of sick pigs is quite resistant to physical and chemical disinfectants. It is not destroyed by heating for thirty minutes at 60° C., but is destroyed by boiling. Common disinfectants, such as carbolic acid and bichloride of mercury, in the ordinary concentrations, exert little effect, while alkaline soap solutions of cresol, such as the compound solution of cresol of the British and United States Pharmacopœias, destroy virus promptly. Blood from pigs sick of swine fever retains its virulence unimpaired for many months if kept in sealed glass bulbs and in a cool, dark place.

Accessory Factors.—The swine fever bacillus (*B. choleræ suis*, *B. suipestifer*) is commonly, though not constantly, found in the blood and tissues of pigs affected with swine fever. It is a small, actively-motile rod which grows readily upon all of the common culture media, and is readily stained by the ordinary aniline dyes, though not by Gram's method. Litmus milk is first rendered acid but later alkaline, without coagulation. It produces gas when grown on glucose broth, but does not change either lactose or saccharose. These fermentative properties serve to distinguish it sharply from *B. typhosus* and *B. coli*. It is markedly pathogenic for guinea-pigs and rabbits, but only moderately so for pigs. The lesions produced in pigs by feeding massive doses or by intravenous injection of pure cultures may resemble those found in pigs that have succumbed to swine fever, but the disease produced by the cultures is not contagious, and pigs that recover do not acquire immunity against swine fever. The common association of the swine fever bacillus with that disease, together with its marked pathogenicity for small animals, and its ability to produce lesions in pigs resembling those found in the natural disease, led to the general acceptance of the conclusion of Salmon and Smith that it was the cause of swine fever. On the other hand, the lack of contagiousness and of immunity following infection served eventually to prove that the swine fever bacillus is not the causal agent.

The swine plague bacillus (*B. suisepticus*) is not uncommonly found in the lung lesions of pigs affected with swine fever. In such cases this infection is strictly secondary to swine fever, though its presence led to much confusion during the earlier investigations of swine diseases. For further description of this micro-organism see SWINE PLAGUE.

Morbid Anatomy.—The anatomical changes caused by swine fever vary greatly in different herds and in different pigs in the same herd, and yet they are usually sufficiently characteristic to enable the practitioner to make a positive diagnosis. The changes in the different tissues may be briefly enumerated as follows :

Skin.—In highly acute cases it is usual to find diffuse purplish blotches on the ears, on the belly and ventral surfaces of the thorax, and between the hind legs. In chronic and subacute cases the skin lesions are generally lacking, and even in very acute cases absence of the skin lesions does not exclude swine fever.

Lungs.—Small reddish spots (ecchymoses), several millimetres in diameter, are frequently found on the surfaces of the lungs, and in addition large reddish or gray areas of solidification (pneumonia) may be found, with or without adhesion of the lungs to the chest wall.

Heart.—The heart is usually normal in appearance except in very acute cases, when irregular areas of blood extravasation may be seen on the outer surface of the heart muscle.

Spleen.—In acute cases the spleen is usually enlarged, soft and dark coloured, while in subacute or chronic cases these lesions are absent and the spleen may be pale in colour and rather smaller than normal.

Kidneys.—The changes in the kidneys caused by swine fever are very commonly present, and as a rule are very characteristic. They consist of punctiform hæmorrhages in the cortex of the kidney, and are best seen after careful removal of the fibrous capsule. These hæmorrhagic points may be one or two mm. in diameter, though they are usually smaller, being for the most part mere dots, and may be overlooked unless a careful examination is made. In well-marked lesions of the kidneys the hæmorrhagic spots of varying size give to the organ an appearance suggesting the speckling of the turkey's egg. The kidney lesions are, as a rule, most marked in acute cases, and may be absent in old chronic cases.

Bladder.—In acute cases, extravasations of blood may be seen in the mucous lining of the bladder.

Stomach.—The changes in the stomach are not, as a rule, characteristic. The mucous lining may be found eroded, and there may be an extensive congestion with some areas of hæmorrhage.

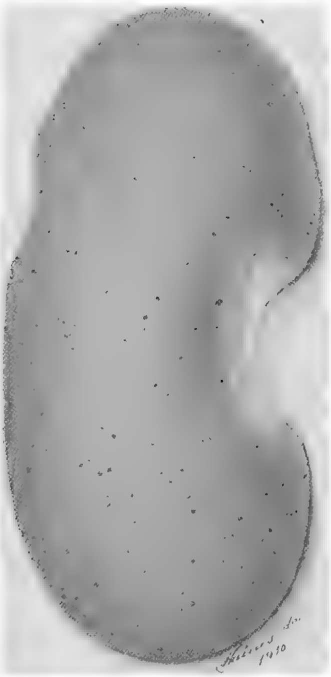

FIG. 28.—Kidney of pig affected with acute swine fever. The numerous small hæmorrhages are shown as dark spots. (Natural size.)

Small Intestines.—The small intestines are not usually the seat of characteristic changes, though at times they may exhibit very marked deviations from the normal. In very acute cases the inner coat and at times the outer coat of the small intestines may appear blood-flecked, thus giving the impression of having been spattered with blood. These are, however, hæmorrhages within the tissue, as they cannot be removed by washing. There may also be found occasionally a diffuse inflammation of the mucous membrane which is characterized by a thickening of the membrane, and the formation of a thick yellowish exudate which is quite adherent to the membrane beneath it.

Large Intestine.—In very acute cases the large intestine may show changes of the same general character as are found in the small intestine, but such cases are rather unusual. In subacute and chronic cases there are found in the mucous membrane the characteristic button-like ulcers which have long been regarded as pathognomonic of swine fever. These ulcers are usually located in the cæcum and the first twelve inches of the colon. In the early stages after the intestine has been opened and flushed out with water, small areas a few millimetres in diameter and yellowish in colour may be seen here and there on the mucosa. When the yellowish exudate is removed from these areas a shallow erosion of the mucous membrane may be seen beneath it. These small erosions are no doubt the beginning of the button-like ulcers which appear later in the disease. The characteristic ulcers of swine fever are round, somewhat raised above the surface of the surrounding healthy membrane, and with more or less distinct concentric rings about discoloured and necrotic though firm centres. These ulcers vary in size from a few millimetres to two or three centimetres in diameter. Their appearance is so striking and so different from the ordinary ulcers with eroded and depressed centres that once observed they cannot well thereafter be mistaken.

Lymphatic Glands.—The lymphatic glands are generally the seat of marked changes, particularly in acute cases of swine fever. At autopsy it is convenient and important to examine the superficial inguinal glands which are found in the tissues just beneath the skin of the belly on either side in the region of the

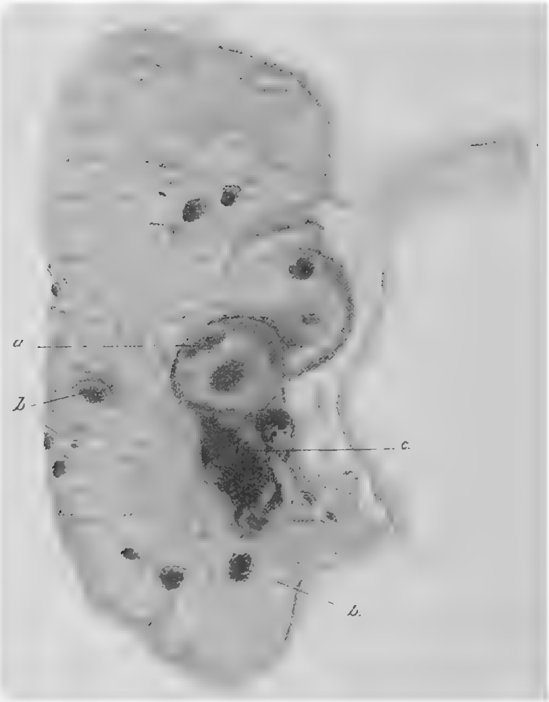

FIG. 29.—Mucous surface of cæcum and part of colon from pig dead of swine fever. The ileo-cæcal valve is swollen and ulcerated. *a*, Ileo-cæcal valve; *b*, "button" ulcers; *c*, large ulcer resulting from coalescence of several "button" ulcers.

flanks. In acute swine fever it is common to find these glands enlarged and red. The red colour is due to the extravasation of blood, which may be so severe as to give the glands the appearance of a dark red blood-clot. In glands that are more slightly affected it can be seen upon section that the hæmorrhage takes place primarily in the cortex. In other lymphatic glands, both of the body and of the viscera, similar changes are likely to be found in acute cases, while in chronic cases these lesions are less marked and may at times even be entirely absent.

To recapitulate: The important diagnostic signs of swine fever to be looked for at autopsy are as follows:

1. Purplish blotches on the skin.
2. Enlarged and hæmorrhagic lymphatic glands.
3. Extravasations of blood on the surfaces of the kidneys, heart and lungs, and on both outer and inner coats of the intestines, stomach and bladder.
4. Enlargement of the spleen in acute cases.
5. Button-like ulcers in the cæcum and colon.

It is rare to find all of the typical lesions in a single pig; in fact there may be individual cases where the autopsy findings leave one in doubt, so that the diagnosis must be made independently of them.

Symptoms.—The symptoms of swine fever, particularly in acute cases, are not distinctive. They are merely such as may be observed in any severe infection. There is high fever (105°-108° F.) from the earliest stages, which falls gradually in case of recovery, while in acute cases which terminate fatally the fever persists until shortly before death, when it drops rapidly to the normal or below. There is loss of appetite, sluggishness, and a disposition to remain sleeping in a dark corner, or to burrow partially under the straw bedding. Pigs at the height of the disease can be aroused and made to move about only after severe prodding. The pig is then seen to be weak, particularly in the hind-quarters; the head and ears droop, and even the casual and inexperienced observer can see that it is quite sick. There is frequently a yellowish, muco-purulent discharge from the eyes. This may be so abundant as to cause the lids to stick together. Diarrhœa, with profuse greenish-yellow discharges, is common in pigs which survive for some weeks; though in the early stages of the disease and in very acute cases this may be entirely absent. Coughing is common, particularly when sick pigs are disturbed. Inspection of a herd of swine shortly after infection with swine fever will generally show that most of the pigs appear to be perfectly well; when they are fed, however, it will be seen that a few do not come to feed, or they may come slowly and eat but little. The sluggish pigs are usually found to have a high fever, and if the temperatures of all animals in the herd are taken, it will usually be found that a number of those that appear to be well have a decided fever (104°-106° F.). The disease spreads rapidly throughout the herd, but careful observers will note that it seems first to attack a few and then from these to spread to the remainder.

Diagnosis.—Inasmuch as there is now available a reliable prophylactic serum for use against swine fever, and as the efficacy of that serum depends in large measure upon its early administration, the diagnosis of the disease has assumed very great importance. A very sharp distinction must be drawn between a "scientific" diagnosis on the one hand and a "practical" diagnosis on the other. By a scientific diagnosis is meant the absolute identification of the disease through a study of the history, symptoms, morbid anatomy and death rate, and through laboratory studies and experimental inoculation of the filtered blood. In the early stages of many outbreaks of swine fever it may be found quite impossible promptly to make a scientifically exact diagnosis. Nevertheless the veterinary practitioner must make a prompt decision, a practical diagnosis, otherwise his services may be of little use to the owner. In reaching a decision the history of the herd and the conditions with respect to swine fever in the locality are important. Sudden and unexplained sickness of a few young pigs should always excite suspicion, and if the sick pigs, as well as others which may be apparently well, exhibit high temperature (105° F.) the veterinarian, in the absence of distinctive signs of other diseases, should kill one of the sick pigs and make examination for lesions commonly seen in swine fever. Discovery of any of the common lesions, such as hæmorrhagic lymphatic glands, or hæmorrhages on the kidneys, heart, lungs, or on either the mucous or serous surfaces of the intestines or bladder, or of diphtheritic or ulcerative changes in the intestinal mucous membranes, is sufficient to warrant a diagnosis of swine fever. Failure to discover at autopsy any of the distinctive lesions of swine fever is on the other hand not sufficient to exclude that disease, and the practitioner in such a case must be governed by his judgement, weighing carefully all of the circumstances surrounding the case, and remembering that early treatment and therefore early diagnosis are essential to success with swine fever.

Differential Diagnosis.—*Swine erysipelas*, in acute form, is very difficult to differentiate from acute swine fever without laboratory studies, which are too slow to be of much service. Violet discoloration of the skin is generally more common and more extensive in swine erysipelas; swelling of the joints of the legs is not infrequent in swine erysipelas, while it is almost never seen in swine fever; hæmorrhages on the serous surfaces are more generally distributed over all of the organs, including the intestines, than in swine fever. In subacute cases of swine erysipelas the peculiar diamond-shaped, red or purplish patches on the skin and the marked lameness caused by the inflamed and swollen hock and knee-joints will usually permit its differentiation from swine fever. It is well to remember that while swine erysipelas may attack pigs of any age, it most commonly affects those from four to nine months of age, while very young pigs and old sows are relatively insusceptible. Swine fever, on the other hand, is commonly fatal to very young pigs, though old sows are usually more resistant.

Tuberculosis should be readily distinguished from swine fever by post-mortem examination. The greyish-yellow areas in the lung and the round firm nodules in the spleen and liver of tuberculous swine are entirely foreign to swine fever.

Swine plague cannot be differentiated from swine fever by a single field examination. Pneumonia is of such frequent occurrence as a complication of swine fever that its presence, even in severe form, is not to be regarded as proof of the absence of swine fever, but more properly as an indication of an outbreak of swine fever complicated with pneumonia. Only in cases where there are found uncomplicated lesions of pneumonia is the practitioner justified in excluding swine fever, and even then such a decision should be made only with hesitation.

Worms frequently infest the lungs and intestines of growing pigs, but the ailments resulting therefrom should be readily distinguished from swine fever. Lung worms cause inflammation of the air-passages in young pigs, and death may follow in severe cases. The diagnosis is made from the fact that quite young pigs only are attacked. There is no fever and a careful examination of the frothy discharge from the mouth which results from frequent coughing will usually disclose the cause, a small worm from one-half to one inch long. Intestinal worms may cause unthriftiness, but fever is lacking, and there are no symptoms of acute disease.

Morbidity and Immunity.—All classes of swine are alike susceptible to swine fever, and yet susceptibility may vary greatly as between herds and between individuals in the same herd. In the United States the actual losses by death in infected herds without treatment probably average from 80 to 90 per cent. It is quite usual to find a small proportion of immune or highly resistant pigs in every herd. As a rule the older members of the herd are found to be more resistant than the young pigs, though quite young pigs farrowed by sows that are actively immune either as a result of artificial immunization or as a result of an attack of the disease, are remarkably resistant. Such pigs are usually entirely immune (a passive immunity derived from the mother) until they are from four to six weeks of age. After that time they lose their immunity, some earlier than others.

An attack of swine fever confers a lasting immunity upon pigs that recover. For this reason farmers in the United States prize such pigs and frequently reserve them for breeding stock.

Prevention and Treatment.—No drug or medicine is known that can be regarded as having any value as a cure or preventive of swine fever. Farmers should therefore be warned against the purchase of drugs or medicines for such use. The only substance known to have any effect is a specially prepared serum which is described below.

Much may be done by swine-growers to prevent the occurrence of disease in their herds. Certainly the most potent factor in the spread of swine fever is the infected pig. Even several days before they exhibit any symptoms whatever, infected pigs may communicate disease to others, and this characteristic infectiousness remains throughout the course of the disease. The virus is disseminated by means of the urine, fæces, eye secretions, and nose secretions. The floors, bedding, and manure in pens or stalls where pigs sick of swine fever are housed become saturated with the virus, which may be carried from one farm to another on the feet of men or animals, or on the wheels of vehicles. After the removal of all sick pigs the virus is gradually destroyed by natural agencies, more quickly in the hot weather of summer than during the winter months. Carcases of pigs which die of swine fever in cold weather may retain the virus in virulent form for months, while in warm weather, when putrefaction is active, it disappears more quickly. Under certain conditions the cured meat from slaughtered animals may contain the virus of swine fever. These facts indicate that farmers may do much to eliminate danger of swine fever by observing the following precautions:

1. Purchase swine for feeding and breeding only from farms and districts free of swine fever. Never purchase from public yards, and do not transport stock in common stock cars or drive over public highways.
2. Locate pig-yards and styes away from streams and public highways and arrange them so that they will not be traversed by men or animals.

3. Quarantine all new stock for three weeks in a remote section of the farm, and admit to the main herd only after careful observation has proven that they are healthy.

4. Maintain a strict quarantine about the pig lots and do not enter pig lots on other farms.

5. Cook all garbage before feeding to pigs.

6. After an outbreak of swine fever see that all carcases are burned, buried deep, or otherwise disposed of so as to prevent reinfection.

7. Unless immunization is practised wait at least three weeks in summer and three months in winter, after removal of the last sick pig, before restocking premises where swine fever has existed.

The above precautions will reduce the danger of infection but will not eliminate it. When swine fever makes its appearance in the immediate vicinity, the herd should either be immunized or the farmer should make arrangements to have immunization carried out immediately following the appearance of symptoms of sickness in the herd.

Preventive Inoculation. *Hyper-immune Serum.*—Pigs that recover from swine fever are thereafter immune against the disease. This immunity is so complete that recovered pigs remain quite well after receiving injections of blood from sick pigs in amounts several thousand times the fatal dose for non-immunes. Observation of these facts led to the development in the United States during the years 1903–1908 of the serum which is now used almost universally for combating swine fever. This serum is prepared as follows:

Pigs, preferably weighing more than 150 pounds and actively immune against swine fever, are injected intravenously with defibrinated blood obtained from pigs sick of swine fever, in the proportion of 5 c.c. for each pound of body weight. The disease-producing blood, or "virus blood," as it is commonly called, may be injected subcutaneously or intraperitoneally, but in that case larger doses of virus blood are required and, particularly when the subcutaneous method is used, the injection is more difficult and is likely to result in the formation of large abscesses due to imperfect and slow absorption. This injection of immunes with large amounts of virus blood is called hyper-immunization.

When the hyper-immunized pig has entirely recovered from any transient ill-effects, and not earlier than ten days after the injection of the virus blood, it is ready for bleeding. The blood may be secured by severing the carotid artery, which results in a large yield of blood; the "hyper-immune" is of course killed as a result. Another method, known as "tail bleeding," is more economical and is used almost universally by the large serum establishments in the United States. The tail bleeding is carried out as follows:

Ten days after hyper-immunization, the serum-producing pig is placed in a suitable crate, and after proper washing and disinfection the end of the tail is chopped off with a very sharp knife or other suitable instrument, care being taken to avoid crushing or bruising the tissues. Enough of the tail must be removed to ensure a good stream of arterial blood, which usually flows from several arteries. It is customary to draw 5 c.c. of blood for each pound weight of the hyper-immune. A ligature is placed around the tail and after proper surgical attention the animal is released, not visibly affected by the bleeding. A second bleeding is made in the same manner after an interval of one week, and may be repeated until a total of three or four bleedings have been made. The pig may then be rehyper-immunized or otherwise disposed of.

The blood from the hyper-immune is defibrinated. In the United States it is the general custom to add to 9 parts of the defibrinated blood 1 part of a 5 per cent aqueous solution of phenol; the finished product thus secured is known as "anti-hog cholera serum," although it is in reality defibrinated blood. It is the cheapest form in which the protective serum can be administered, and although it is not sterile and may at times produce abscesses, the successful use of it on millions of pigs is sufficient proof of its value.

Before the addition of phenol the blood-cells may be removed by simple centrifugalization, whereby about 50 per cent of relatively clear serum is separated, or there may be applied the method recently described by Dorset and Henley of adding to the defibrinated blood a small amount of an extract of the common white navy bean, which causes the red blood-cells to agglutinate, and of common salt, which causes them to contract. When the mixture—defibrinated blood, bean extract, salt—is centrifuged the cells are packed into a firm jelly-like mass, from which a perfectly clear serum may be readily removed by pouring. The yield of serum is usually 75-80 per cent of the volume of the defibrinated blood.

After the serum has been collected in large amounts (80 to 100 litres) it is mixed, bottled, and subjected to test for potency. The test is usually made by injecting eight pigs weighing from 40 to 90 pounds each subcutaneously with 2 c.c. of virus blood; simultaneously two of these pigs receive each 15 c.c. of the serum, two receive 20 c.c. of serum, and two receive 25 c.c. The remaining pigs receive no serum but serve as controls. If the controls develop swine fever while all of those that receive serum remain well, the serum is ready for the market.

If those which receive 15 c.c. of serum become sick while those which got the larger doses remain well, the serum is marketed only under an increased dose.

While this test does not give precise information as to the relative potency of different sera, it serves to eliminate sera that are below the required standard, and furthermore may give warning in case of the presence of contaminating micro-organisms.

Methods of applying Hyper-immune Serum.—The swine fever hyper-immune serum, or "anti-hog cholera serum," of the United States may be used in practice in several different ways: (1) The serum alone is injected subcutaneously—this is known as the "serum alone method"; (2) The serum is injected subcutaneously, and at the same time, on the opposite side of the body, a small amount of virus blood is injected subcutaneously—this is known as the "simultaneous method"; (3) the serum alone is injected and, after an interval of ten days to two weeks, the simultaneous injection of serum and virus is given.

The last-named method of procedure is expensive and is little used.

The Serum Alone Method.—The serum alone is used either to protect healthy pigs or to treat those already infected. In common with other anti-sera, its value is chiefly as an immunizing agent, though in many cases, particularly in the very early stages of the disease, it has distinct curative effect. The passive immunity which follows the injection of serum alone is of short duration, lasting usually from three to six weeks, though in some cases it seems to persist for several months. The serum alone, if free of contamination, cannot possibly do harm to the treated pigs; its only defect lies in the relatively short period of immunity that follows its use.

The Simultaneous Method.—This method is used exclusively to protect healthy pigs. Its advantages and disadvantages are directly opposed to those of the serum alone method, for, while it produces a firm and lasting immunity, it involves a certain element of danger owing to the use of the virus. Except in the case of very young pigs from immune mothers the immunity appears to last for life. It is important to note that successful immunization by this method is dependent upon the use of virus of full virulence and of ample doses of potent serum. No attempt should be made to titrate serum against virus so as to use barely enough serum to secure protection. Such a system leads almost certainly to "vaccination disease," which should always be avoided. The two important elements in simultaneous treatment, as already indicated, are a full dose of active virus with an excessive dose of serum. Excess of serum appears in no wise to shorten immunity, while it does afford the protection against loss from treatment which is essential.

In the United States the general preference of farmers is for the simultaneous method. It has now been in use in many of the States for several years and, coincidently with increased application of the simultaneous method, there has been a steady decline in prevalence of hog cholera.

Practical Considerations.—The veterinary practitioner is frequently called upon to decide what method of treatment shall be applied. In considering this question, herds may be placed in three classes: (1) Healthy herds not in an infected district; (2) Healthy herds in districts where swine fever is prevalent; (3) Infected herds.

(1) *Healthy Herds not in an Infected District.*—It is well known that swine fever does not spread merely by extension from farm to farm, but that the disease may suddenly break out in localities far removed from any known centre of infection. One cannot, therefore, be sure of safety merely because of remoteness from existing disease. However, the serum is expensive and the veterinarian must weigh the likelihood of infection against the cost of immunization. Consideration must also be given to the possibility of infection resulting from the simultaneous inoculation. In the absence of near-by infection it would manifestly be unwise to use the serum alone, as the transient immunity resulting therefrom could hardly compensate the owner for the cost. Accordingly in herds of this class the serum alone is of doubtful utility, and the simultaneous inoculation should be advised only after full explanation has been made to the owner of the danger involved, and after steps have been taken to control promptly any infection that may result from the inoculation.

(2) *Healthy Herds in Districts where Swine Fever is prevalent.*—Here the danger is imminent, and healthy herds should be immunized unless it is possible to arrange for immediate treatment following the first symptoms of disease in the herd. Even in infected districts there is always, with the exercise of proper care, the chance of avoiding infection, but the running of such a risk is advisable only when inoculation can be immediately applied upon the appearance of the first signs of sickness. The serum alone may afford sufficient protection in communities where the centres of infection are promptly cleaned up, but failing assurance in that respect, the simultaneous inoculation is recommended, provided it be applied by a competent veterinarian, who is able to keep the herd under observation from day to day. The veterinarian should, in such cases, bear in

mind the importance of preventing the development of any sickness among the treated pigs by administering a liberal dose of serum.

(3) *Infected Herds*.—The degree of success attained in the treatment of infected herds is in direct relation to the time of inoculation. The earlier the inoculation the greater the success. On infected farms there seems to be no objection to the use of the simultaneous inoculation. The infection is already present, and there can be no question of the danger of setting up an additional centre. It is true that the serum alone is usually quite effective in stopping losses during an outbreak, but there is then greater likelihood of a recurrence of the disease among the pigs which are unaffected at the time of inoculation than when the simultaneous method is used upon the healthy pigs. In all herds where disease exists the temperature of each pig should be taken and recorded before treatment. All pigs, regardless of other symptoms, that exhibit a temperature exceeding 104° F. should be given a liberal dose of the serum alone, while those with lower temperatures and without other symptoms should be given the simultaneous inoculation. The subjoined dose table indicates roughly the doses of serum and virus that have been found effective.

Doses of Serum and Virus in Simultaneous Inoculation of Healthy Pigs

Weight of Pigs.	Dose of Serum.	Dose of Virus.
Below 10 pounds	10 cub. cent.	
10-15 ,,	15 cub. cent.	¼ cub. cent.
20-30 ,,	20-25 cub. cent.	½ ,, ,,
40-75 ,,	30 cub. cent.	1 ,, ,,
100-150 ,,	40-60 cub. cent.	2 ,, ,,
175 pounds and over	80 cub. cent.	2 ,, ,,

If the herd is infected, the dose of serum should be slightly increased for all apparently healthy pigs, and all those showing high temperatures or other evidence of disease should receive at least a dose and a half of serum and no virus.

It is important to remember that in diseased herds early treatment is essential to success, and that liberal use of serum will give results which will amply compensate the owner for the increased cost of treatment.

M. D.

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SWINE PLAGUE [1]

Synonyms.—Pig pneumonia; Pasteurellose du porc (*Fr.*), Schweineseuche (*Ger.*).

Definition.—A sporadic disease of swine characterized by pneumonia and caused by *Bacillus suisepticus* (Swine plague bacillus).

Swine plague was first described by Loeffler as an independent disease of swine. Salmon and Smith, working contemporaneously in the United States, differentiated swine plague sharply from hog cholera and seemed to show conclusively that in the United States there existed two great diseases of swine, hog cholera and swine plague, which occurred independently of each other and also as mixed infections of the two diseases. This view prevailed quite generally for more than twenty-five years, the diagnosis in doubtful cases being based finally upon the bacteriological findings. If the bacteriologist reported the swine fever bacillus, the disease was swine fever; if he reported the swine plague bacillus, it was swine plague, while recovery of both micro-organisms established the existence of a mixed infection of the two diseases. In recent years, since the discovery of the filtrable virus of swine fever, our conceptions of swine plague as well as of swine fever have been altered. Few if any authorities now believe that swine plague exists as an independent epizoötic of swine. It occurs rather in the form of sporadic pneumonia affecting a few or perhaps at times a considerable number of pigs in a herd, and is encountered not infrequently as a complication secondary to swine fever. As a menace to swine-raising, swine plague is of quite minor importance as compared with swine fever.

Swine plague, or swine pneumonia as it might more properly be called, is caused by a small bacillus, known as the swine plague bacillus or *Bacillus suisepticus*. This micro-organism stains well by Gram's method, is non-motile, and does not produce gas when cultivated on media containing the common sugars. It is usually of marked virulence for rabbits and guinea-pigs. Small doses of virulent strains of the pure culture may cause the death of rabbits within eighteen hours. For pigs the pure culture is usually not markedly pathogenic after subcutaneous injection.

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[1] See also Hæmorrhagic Septicæmia of Swine, p. 110.

The *symptoms* of swine plague are not distinctive, and the lesions found at autopsy are merely those of pneumonia. It is true that many authors ascribe various other pathological changes to this disease, but in such instances the possibility of concurrent swine fever infection does not seem to be eliminated.

The swine plague bacillus is a normal inhabitant of the bodies of healthy pigs. From this it follows that prevention of this disease is to be gained through maintenance of the health and vigour of herds of pigs by proper feeding and sanitation, and by protection from exposure and provision of dry and warm sleeping-places in the winter. In Germany polyvalent serums and vaccines have been recommended, but their value is not regarded as definitely established.

M. D.

SWINE ERYSIPELAS

Synonyms.—Red soldier; the purples; pig measles; Rouget du porc (*Fr.*); Schweinerettlauf (*Ger.*); mal rosso (*Ital.*).

Definition.—Swine erysipelas is an infectious disease of swine, caused by the swine erysipelas bacillus and characterized by high fever, reddish or purplish patches on the skin, and hæmorrhages on the surface of the viscera in acute cases, and by general unthriftiness, difficulty in breathing, and lameness in chronic cases.

History and Distribution.—Swine erysipelas has no doubt existed in Continental Europe for many years. For a long time before its cause was known it was recognized as an independent disease, and as early as 1882 Pasteur and Thuillier prepared a vaccine for use against it. In 1885 Lœffler discovered the bacillus of swine erysipelas and thereby supplied an accurate means for distinguishing this disease from other infections of swine.

In recent years swine erysipelas has seemed to increase rather than diminish in Continental Europe. It exists in all of the large swine-raising districts of Europe and at times assumes the character of an epizoötic, sweeping over large areas and causing great losses. The chronic form exists in Great Britain, and from time to time the disease there also becomes more prevalent and more acute in character. Swine erysipelas is not known in the United States.

Etiology.—The cause of the disease is the bacillus of swine erysipelas (*B. rhusiopathiæ suis*). This micro-organism is a very slender rod which, in acute cases, is found in the blood, particularly in the large phagocytic white blood-cells, also in the spleen, kidneys, the purple lesions on the skin, and in the fæces and urine. In chronic cases the bacilli are not so widely distributed, but may be found in the vegetative growths on the heart-valve and also in the spleen. It is stained well by Gram's method as well as by most of the ordinary stains employed in the laboratory. It is not motile. It grows in the laboratory best in the absence of air, and in the case of deep stab cultures on gelatine grows in a very characteristic form, the growth resembling the hairs of a test-tube brush radiating from a central stem. Mice and pigeons are very susceptible to inoculations of the pure culture, while most other animals are quite resistant. Men may be infected through accidental wounds, but the disease usually is localized in the region of the point of infection.

Morbid Anatomy.—The carcases of swine that have died of acute swine erysipelas generally show the following changes from normal: The skin shows red or purple splotches which may at times be so extensive as to cover the entire surface of the body; the lungs are congested, and hæmorrhages may be seen on both the outer and inner surfaces of the heart-muscle. The spleen is enlarged and rather dark in colour, while the kidneys generally show small circumscribed hæmorrhages dotted over the surface. The outer coverings of the stomach, intestines, and bladder exhibit hæmorrhages, while the inner surface of the small intestine, in particular, may show signs of inflammation but is not ulcerated. The lymphatic glands are swollen and red.

After death from chronic swine erysipelas the hæmorrhages, which are so constant and striking in acute cases, are much less prominent and may be lacking entirely. The most constant and striking lesion consists of the cauliflower-like growths that are found on the normally smoother valves of the heart. There is usually an œdema of the lungs, and the inner coat of the intestines may show evidence of chronic inflammation.

Symptoms.—It is customary to speak of three forms of swine erysipelas: (1) Mild; (2) Acute; and (3) Chronic.

The mild form is rarely fatal, the deaths occurring only among those that develop the chronic disease following a mild attack. The symptoms come on suddenly, there is high fever, complete loss of appetite, and marked dullness. On the skin, especially over the thorax, neck, back, and on the outside of the thighs, rather distinct red or purplish areas, which not infrequently are square or diamond shaped and are raised above the surrounding skin, become visible a day or two after the onset of the disease. The hock and knee-joints may be swollen and painful. As a rule the symptoms abate in four or five days, and the pig makes a rapid recovery.

The acute form in the beginning is much like the mild form, there is great prostration, very high fever, and complete loss of appetite. The discoloration of the skin, which appears early, is more extensive than in the mild cases, the red patches become confluent, and the entire surface of the skin may become red or purple. Diarrhœa may follow the constipation which practically always exists in the early stages. In fatal cases death may take place within twenty-four hours or the pig may live eight or nine days.

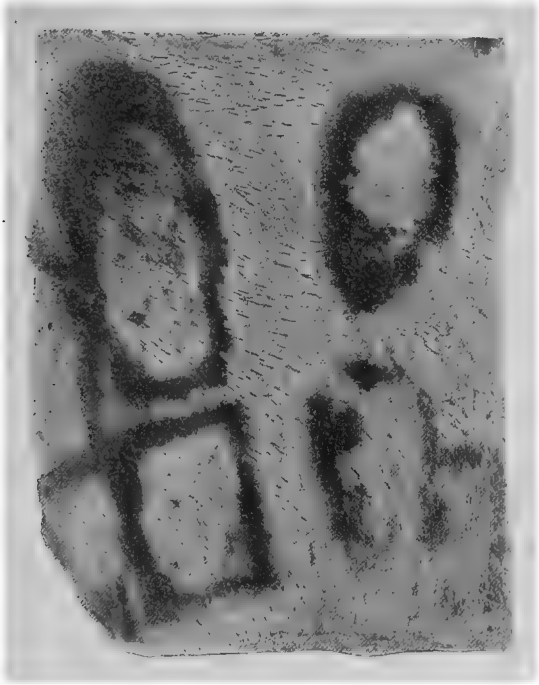

Fig. 30.—Skin of pig with characteristic markings in swine erysipelas.

The chronic form is to be regarded as a sequel of a mild or an acute attack. There is little or no fever, the pig is unthrifty, and breathing is difficult. There is usually a cough, and discolorations of the skin may be seen which become more pronounced and purplish as the disease progresses. The sick pig is disinclined to move about and rests usually on the sternum and elbows. Recovery seldom, if ever, takes place.

Differential Diagnosis.—In countries where both swine fever and swine erysipelas occur it is important and frequently difficult to differentiate the one disease from the other. In acute swine erysipelas fat pigs of medium age are most susceptible, while quite young pigs are relatively immune. There is no such marked difference in susceptibility to swine fever, as the very young pigs are relatively even more susceptible than the older stock. In swine erysipelas the skin discolorations may assume the characteristic diamond shape, and the hæmorrhages on the internal organs are generally more marked on the serous than on the mucous membranes. A point of distinction that is of prime importance is the fact that swine erysipelas never causes ulceration of the intestines, while in swine fever, even in acute cases, it is of common occurrence.

Prevention and Treatment.—To check the spread of the disease the usual methods of quarantine of premises, disinfection of infected pens and pen litter, and prompt disposal of carcases of dead pigs by burying or burning, should be practised. Effective serums and vaccines are available for use against swine erysipelas, and they should be used in preference to treatment by drugs or chemicals.

The original vaccine against swine erysipelas was prepared by Pasteur. The virus is attenuated by passage through rabbits, and cultures made from the rabbits are used to inject pigs. This method has been extensively and successfully used in Hungary, and to a less extent in France, Russia, and Germany. It involves a certain danger of loss from vaccination, reaching as high as 10 per cent in some cases. More recently Leclainche in France and Lorenz in Germany have each produced an immune serum by injecting horses repeatedly with cultures of swine erysipelas bacilli. Blood serum from such horses has the power to protect healthy animals against the natural infection as well as against pure cultures. This serum may exert some curative action in the case of sick animals if given in liberal and repeated doses. Its chief value lies, however, in its protective action. It may be used alone, in which case it protects for two or three weeks. The serum is most usually applied in combination with virulent swine erysipelas bacilli. The pigs to be treated are injected with serum and bacilli simultaneously, either mixed together (Leclainche) or given separately, on opposite sides of the body (Lorenz). From ten to fifteen days after this simultaneous inoculation the pigs are given an injection of the virulent cultures alone. In treating herds where infection already exists it is recommended to give all pigs, sick and well, a dose of serum alone; ten days later the regular vaccination with serum and culture and culture alone is carried out in the manner already described.

M. D.

BRAXY

Brasot, Bradsot, Black Disease

Braxy is a contagious disease affecting sheep, characterized by sudden attack, abdominal pains, and very rapid course. It occurs mainly along the west coasts of Scotland and Ireland, and England (Northumberland and Cornwall), Iceland, Scandinavia, and Northern Germany. **Black Disease,** which closely resembles braxy in its clinical features, and is regarded by Dodd as probably identical with braxy, is met with in Australia (New South Wales and Victoria). Owing to its rapid course braxy has frequently been confused with anthrax, malignant œdema, black-quarter, and louping-ill.

The outbreaks of braxy occur chiefly in the late autumn and early winter, October to January, and cases are seldom met with after January. In the case of black disease in Australia the seasonal occurrence is from February (late summer) until May (early winter). Sheep are frequently found dead of braxy on the morning after a frosty night. First-year lambs after weaning are the most susceptible to the disease. Yearlings are less susceptible, while third-year sheep are almost immune. This resistance, owing to advancing age, is probably due to the sheep having been affected with a mild attack when young, from which recovery occurred leaving a certain degree of immunity. In Australia Dodd found that there was no special age at which the disease occurred, and in some outbreaks he observed the principal mortality amongst adult sheep, and suggested that it may be because the young animals were not so exposed to infection. On the other hand, he has observed young lambs from six to twelve weeks old dying from black disease. Males and females appear to be equally susceptible, though one often finds it asserted that males are more resistant than females.

Etiology.—Braxy is commonly regarded as being due to an organism first described by Nielsen and called the bacillus of braxy (*B. gastromycosis ovis*). The same organism was also described by Hamilton. It is a rod-shaped bacillus from 2 to 6 microns long and 1 micron thick. When the bacillus sporulates, the spore distends the bacillus in the centre or at the end. It is an aerobic organism, a gas producer, and liquefies gelatine. The organism is found in the gastric and intestinal wall, and in the various tissues and body cavities, after death. There is disposition in certain quarters to doubt the specificity of this organism for braxy, it being suggested that the organism is a putrefactive organism which rapidly invades the tissues at the point of death or immediately after the death of the animal. Miessner failed, as a rule, to demonstrate the organism in fresh carcases of animals dead of the disease or which were killed shortly before the fatal termination.

In the case of black disease Dodd has been unable to demonstrate this bacillus with any degree of consistency at or before death in the blood-tissues or exudates of deceased animals. He says: "The instances in which bacteria have been demonstrated to have been present before death are too few when compared with those in which none were found to permit of any generalization with regard to their etiological importance. In spite of this, however, it is quite possible that the cause is bacterial but that the organisms are confined to the lesions or to the intestinal tract whence a powerful toxin is given off."

The period of incubation in naturally occurring cases is not determined, but experimental inoculation of the so-called braxy bacillus appears to cause the disease in from 2 to 3 days. The course of the disease is very rapid, in the majority of cases so rapid that symptoms are unobserved, the animal being found dead. In other cases, where the affected sheep are under close observation, the course may extend from 15 to 20 hours. The mortality in naturally occurring cases cannot be made out owing to the probability of a number of mild cases passing unobserved. In those cases where symptoms are observed, the disease is almost invariably fatal. The mortality amongst a flock of young sheep in an affected area may be up to 30 or 50 per cent.

Symptoms.—The symptoms of braxy occur very suddenly and develop rapidly, so that in a very large number of cases it is fatal before any symptoms have been observed. In other cases, where careful observation has been kept, the sheep is found to gradually lag behind the rest of the flock until it is unable to keep up with them and is left behind. There is some nervousness and a quick, short step, appetite is lost, and rumination ceases. The sheep is restless, lying down and getting up constantly and grinding its teeth. The pulse is weak and frequent, and respiration quick and laboured. The temperature may be anything from 104° to 107° F. Diarrhœa is sometimes observed. When standing the head is hung down, the back is arched, and the sheep shows signs of a cramping abdominal pain. It soon falls over and rapidly becomes comatose and death supervenes.

Post-mortem Appearances.—The carcase rapidly undergoes decomposition and becomes tympanitic, the skin undergoing the typical green discoloration of sheep dead from any cause if left uneviscerated. Similarly, many of the conditions described in autopsies are due to post-mortem decomposition. If a post-mortem examination be made on an animal killed at the point of death or as soon as dead, before putrefactive changes have taken place, the principal lesions will be found in the fourth stomach and the small intestines. In the former position there is usually a marked congestion, especially at the pyloric end of the stomach, while the small intestines, particularly the ileum and jejunum, show a similar congestion, sometimes very severe, at other times less intense. In rare cases hæmorrhage in the pyloric end of the stomach and intestines has been recorded. The abdominal lymphatic glands are somewhat enlarged and œdematous. The peritoneal, pleural, and pericardial cavities often contain a varying quantity of blood-tinged serous fluid, and a puff of gas frequently escapes on puncture of the pericardium. The organs rapidly become spongy and friable, changes which are probably due to post-mortem invasion.

Treatment.—No curative treatment of any value is known.

Prevention.—The carcase of a dead sheep should be properly disposed of either by cremation or deep burial in order to avoid the spread of organisms from the discharges. It is inadvisable also to salve the skin and fleece owing to the possibility of distributing infection by that means. All first-year sheep should be removed from the affected districts in the early autumn and kept away until after January, when they may be returned to their former pastures at the expiration of the braxy season.

Preventive measures are sometimes adopted, consisting mainly of the injections of vaccines prepared from the so-called braxy bacillus, or of serum from horses hyperimmunized with virulent cultures of the organism, or a mixture of both. By these methods it is stated that mortality has been reduced to less than 2 per cent. Another vaccine has been prepared from the kidneys of sheep dead of braxy in a similar manner to Arloing's method of preparing black-leg vaccine from affected muscle-tissue. Good results have been claimed for this method.

The method of prevention adopted in Scotland consists in the administration by the mouth of organisms presumed to have been attenuated by their passage through the alimentary tract of swine (the drenching with emulsions of the dung of pigs that have grazed for several days over a braxy-affected pasture).

G. H. W.

LOUPING-ILL

Tremblings ; Ovine Chorea

Louping-ill is an infective disease of sheep which is transmitted by ticks. Occasional cases have also been said to occur in swine and cattle. It is apparently confined to Scotland, the North of England, and the North-West of Ireland. Louping-ill infected localities may be sharply marked off from clean ground, no doubt due to the conditions in the former being more favourable to the development of the ticks.

Etiology.—The tick responsible for the transmission of this disease is the *Ixodes ricinus*, which may inoculate the disease, according to Stockman's researches, both in the larval and in the adult stages. When inoculated by larvæ the latter are developed from eggs deposited by adult females which have fed on louping-ill affected sheep. When inoculated by adults the ticks have become infected in the nymphal stage by engorging on louping-ill affected sheep. It would appear that a gross infestation is necessary for the transmission by larvæ, but not in the case of transmission by adults.

The causal parasite inoculated by the ticks is not positively made out, but it seems more than probable that it is a protozoan parasite; and certain bodies which may ultimately prove to be protozoa have been described by Stockman in mono-nuclear leucocytes in the blood and œdematous lymphatic glands. Whatever it be, the virus is not filtrable, and virulent material when perfectly fresh is free from bacteria. Various bacteria have been described as causing louping-ill, particularly a sporulating organism which is frequently found in the peritoneum on post-mortem examination of animals dead of louping-ill. This organism is probably a putrefactive organism.

Outbreaks of louping-ill are usually met with at two seasons of the year, viz. in the spring from April to June, and later in the autumn, chiefly September. These periods correspond exactly with the first and second crops of ticks for the year, and this seasonal occurrence affords strong support to the tick transmission statement. The rarity of louping-ill during the months of July and August is therefore probably due to the absence or scarcity of ticks and not to any special bactericidal action of sheeps' blood at that time.

Symptoms.—Acute and subacute forms may be recognized. In the *acute form* of louping-ill symptoms are shown from 4 to 6 days after infestation with ticks. The sheep appears to be uneasy, frequently lying down and getting up again, the temperature ranging from 104° to 107° F. during the next 8 or 9 days. The sheep is more than usually timid and nervous, with trembling or twitching of the muscles of the jaw and frothing at the mouth and lack of co-ordination. In the advanced condition the sheep may be found completely comatose, with its head drawn round towards the shoulder.

In the *subacute* form the symptoms of mild cases may amount to little more than slight indisposition. In others the affection is more severe, giving rise to dulness, reeling gait, muscular spasms, and convulsions. These may be followed by stupor or by paralysis of both fore and hind limbs while retaining consciousness. In this latter condition the sheep may lie for weeks feeding with variable appetite, but the attack usually terminates in death. Recovery from one attack of louping-ill confers a considerable degree of resistance, but not absolute immunity. Stockman's researches tend to show that recovered animals do not remain carriers of infection.

Lesions.—According to Stockman the only constant lesions in louping-ill are found in the lymphatic glands, particularly of the abdominal cavity, notably those of the lumbar region. These glands are found to be swollen and œdematous, very rarely congested. There is also frequently a superficial hæmorrhage in some part of the brain, and the anterior part of the spinal cord. On microscopic examination of preparations made from the glands small bodies, either round, oval, pear-shaped, or bacillary, may be found in the mono-nuclear cells, and similar bodies may be found in fresh blood in acute cases. There appears to be no alteration in the number or character of the red corpuscles. Microscopic examination of the brain and spinal cord demonstrates congestion of the small vessels, with or without hæmorrhages into the surrounding tissues; at other times a more or less intense inflammation with the migration of leucocytes through the vessel walls into the sheaths and nerve tissues. The central canal of the spinal cord may contain a small amount of jelly-like substance. Various other lesions that have been described are ascribable to post-mortem changes.

Treatment.—No curative measures of any service are known. Stockman found various arsenical preparations and trypanblue to be equally useless.

Preventive measures may be of some service. As the disease has proved to be a tick infection, the best results are likely to be obtained by attempting to eradicate those parasites. Rough herbage and withered grass and brushwood should be burnt off during the winter months. An attempt may also be made to starve out the ticks, which necessitates the leaving of the pasture free of sheep for over a year, since *Ixodes ricinus* can live over a year under natural conditions. The land may at the same time be generously dressed with salt or lime.

Another method is to graze adult or immunized sheep over the pastures during louping-ill periods in order to collect the ticks, and to dip such sheep every five days during that period with some effective parasiticide.

Attacks of the disease artificially produced by the inoculation of virulent blood are less severe than natural attacks, and result in recovery with some degree of resistance. A method of immunization may possibly be developed on these lines.

G. H. W.

SCRAPIE

Scrapie is a disease peculiar to sheep and characterized by intense itchiness and certain nervous symptoms. The disease exists in the extreme North of England and in the South of Scotland, and also in certain parts of France and Germany. Careful researches appear to show that it has been in existence in Great Britain for over a century, although it is only recently that attention of veterinary surgeons has been called to it. All breeds appear to be susceptible, though the disease is most commonly met with in Cheviots, Scotch Black Face, and Border Leicester sheep. The sexes appear to be equally susceptible. The common age at which sheep are affected is between 18 months and 2 years, although very rare cases have been reported in lambs born of badly infected ewes. If it really does occur in lambs it must be exceptionally rare.

In the present stage of our knowledge of this disease it is quite impossible to fix the period of incubation. In the event of the disease being congenital, and the fact that authentic cases are rarely or never seen before a sheep is from 18 to 20 months old, it would follow that the period of incubation must extend over that time. On the other hand, if the method of infection is by copulation, since the ewes generally show symptoms about two months before lambing, it would follow that the period of incubation would be approximately three months.

Etiology.—The cause of scrapie is not known, nor is the method of spread well understood. It appears possible, however, that the method of transmission is by copulation and also by contact with an infected animal during the period of incubation. It is also possible that it may be transmitted by inter-uterine infection, so that lambs may become infected either before birth or by contact with infected mothers after birth. Cases have been recorded by McFadyean and Stockman of the introduction of the disease by a ram into a previously sound flock, and the former also records a case in which a ram bred in a sound flock was used for service in a flock known to be infected with scrapie, and subsequently developed the disease. McGowan is of opinion that the disease is due to a heavy infestation with sarcocysts in the muscles, and which may be passed on by the ewe to her progeny. This view has not yet been corroborated.

Symptoms.—The symptoms of scrapie are partly cutaneous and partly nervous.

The *cutaneous symptoms* are first developed, and consist of a marked itchiness of the skin, the sheep rubbing itself against any suitable objects and showing gratification if any part of the skin is scratched with the fingers. In consequence of the rubbing, the fleece soon becomes ruffled and damaged, the irritation increases in intensity and extends to the legs and head. The constant scratching and nibbling causes a very great loss of condition and sometimes results in mechanical injuries to the skin. In chronic cases almost the whole fleece may be shed and a new growth of wool may take place. The skin symptoms have attracted more attention in this country than the nervous symptoms.

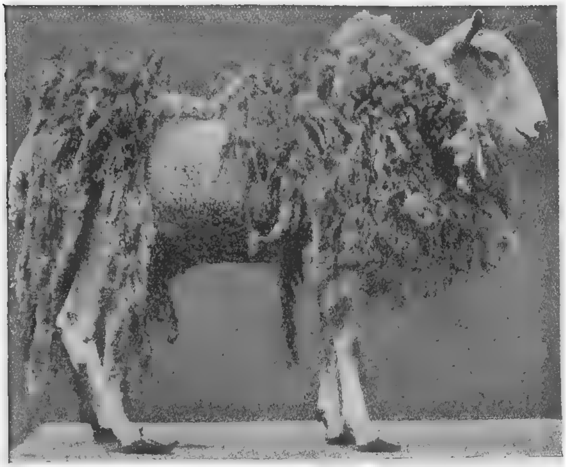

FIG. 31.—Scrapie. (From the *Journal of Comparative Pathology and Therapeutics.*)

Nervous Symptoms.—In some cases these are much more pronounced than the pruritus. The affected sheep are noticed to raise their heads with ears inclined backwards as though listening intently. There is a peculiar high action of the fore limbs when running, and when standing the body is often in a state of tremor or marked shivering. (Owing to these peculiar nervous symptoms the German name "Trotting Disease" and French "The Trembles" have been applied, and it seems possible that this condition may be responsible for some of the cases exhibiting nervous symptoms usually described as louping-ill in Scotland.) When startled, epileptiform seizures are sometimes set up, while at other times no convulsions are produced. McFadyean records a case in which a ewe, apparently perfectly healthy, constantly fell over on her side when a dog was sent round the flock. This ewe subsequently became paralysed and died 30 days later. Death usually occurs from general exhaustion. On post-mortem examination no lesions have been discovered which are specially indicative of scrapie.

Treatment.—No curative measures appear to be of any service. McFadyean is of opinion that the only certain method of eradicating the disease is to dispose of the whole of the sheep, young and old, by fattening for the butcher, and to restock with ewes and rams obtained from sound flocks. When new stock is introduced a guarantee should be obtained that the animals are from flocks known positively to be free from the disease. As it is also suggested that as the disease may be either congenital or contracted during the first few months after birth, and to show itself before the animal attains the age of two years, it would be a sound plan on purchasing rams not to put them with the ewes until the former are in their third year. G. H. W.

HEARTWATER

Heartwater is a specific tick-transmitted disease of cattle, sheep, and goats, caused by an ultravisible virus, and characterized post mortem by an accumulation of liquid in the pericardial sac, a striking lesion from which the popular name has been derived.

Various other names have been applied to the disease by South African farmers, particularly "Boschziekte" (bush-sickness), Veld-sickness, and Inapunga, but the conditions passing under such popular names cannot as a rule be regarded as distinct pathological entities. As has been pointed out by Theiler, a case of Heartwater in which the best-known and most characteristic post-mortem lesion was wanting would probably be diagnosed as Boschziekte or Galziekte, or possibly as "Dronk Galziekte" (drunken gall-sickness), although "Galziekte" could not properly be given as a synonym for Heartwater, as the name is extended to all cases of disease (particularly in cattle, but also in sheep) showing a particular train of symptoms, and the majority of bovine cases so described are, of course, due to infection with *Anaplasma marginale.*

Similarly a case of some other disease, in which post-mortem examination revealed any very noticeable amount of liquid in the pericardial sac, would often be ascribed to Heartwater, although there might be few or no other features in the case to suggest such a connection. Theiler records that cases of East Coast Fever have often been mistaken for Heartwater (and East Coast Fever is still very frequently diagnosed by farmers as Gall-sickness), and many cases of parasitic invasion, leading to emaciation and the presence of transudates in the serous cavities, have been attributed to the same cause.

History.—The disease has probably existed for a long time in such territories as Zululand, Swaziland, and the Transvaal low veld, and Theiler remarks that it has long been recognized by the Transvaal Boers. As regards the Eastern Province of Cape Colony, the disease would appear to be of more recent introduction. Spreull states that " bont " ticks were first seen in the Lower Albany district in 1840, and that small stock began to do badly about 1860, while Lounsbury quotes a report from the magistrate at East London in 1875, in which it was stated that sheep and calves were becoming increasingly difficult to rear in certain parts of his district. Of recent years the distribution of the disease has been considerably affected by the constant dipping of cattle, and the disease has ceased to be of any great importance in many parts of South Africa in which it was formerly very prevalent. On the other hand the very considerable amount of settlement and development which has been occurring during the past few years (and is still going on rapidly) in the low veld areas of the Transvaal, Zululand, and Swaziland, has caused the disease to acquire renewed importance.

Geographical Distribution.—In the Union of South Africa the disease is, or has been, more or less widespread in the " low " or " bush " veld of the Transvaal, and in the low-lying warmer parts of the eastern coastal belt, extending through the territories of Zululand, Natal, the Transkei, and the Eastern Province of Cape Colony.

Local Distribution.—The disease is contracted only in the warm, low-lying " bush-veld," and only in those parts with actual bush ; the local distribution is necessarily dependent on that of the transmitting agent, the bont tick.

The disease is more prevalent, and appears to be more virulent, during the summer months.

Animals Affected.—The disease particularly attacks sheep and goats, and in these animals its symptoms and lesions are most characteristically shown. Angora goats are highly susceptible, and merino sheep also contract the disease very severely ; merino crosses with less susceptible breeds show more resistance.

The common Kaffir goat and the fat-tailed sheep are found in the Heartwater areas, and probably over a long course of time have become more or less immune (Theiler). It appears, however, that less resistance may be shown by such animals which have been bred for generations in districts quite free from Heartwater, but they never show the great susceptibility exhibited by the breeds previously mentioned. Persian sheep thrive in Heartwater areas, and, although not absolutely immune, are very resistant ; in the great majority of cases they merely pass through a febrile reaction, during which the blood is infective for susceptible animals, either by direct inoculation or through the agency of ticks.

As regards cattle, Theiler records that the Transvaal Boers long ago recognized a bovine disease as identical with the Heartwater of small stock, but the connection was not so well recognized in some other areas. Edington first succeeded in inoculating the disease to cattle with virulent goat blood, and Lounsbury transmitted the disease to cattle with infected ticks, while Theiler (*Annual Report*, 1903–4) has given a full and very careful study of a number of cases, both experimental and naturally contracted, and has shown that Heartwater may be a very fatal disease of cattle.

All attempts to convey the disease to horses, both by inoculation and by infestation with infected ticks, have failed.

In all the susceptible species and breeds young animals contract the disease more easily and more severely than adults.

Ætiology.—The disease is caused by an organism or virus which is present in the blood during an attack, as can readily be proved by subcutaneous (and still more certainly by intravenous) inoculation into a susceptible animal. The organism has not been demonstrated microscopically, nor has it been possible up to the present time to cultivate it *in vitro*, and although in various experiments it has failed to pass through Berkefeld and Chamberland filters, the organism is presumed to be ultravisible. Inoculation of pericardial liquid may produce the disease, but it frequently fails to do so. The virus cannot be preserved by any known method, and blood drawn from a sick animal retains its virulence only for about forty-eight hours. Edington claimed to have shown that Heartwater and African Horse-sickness were the same disease, or different phases of one disease, but this view was strongly combated by Theiler and Stockman, who were able to bring forward very convincing reasons for not accepting such an hypothesis.

Natural Mode of Transmission.—Lounsbury transmitted the disease to goats and sheep, and later to cattle, by means of infected " bont " ticks, and he failed to transmit the disease with any other species of tick. The results of his valuable work on this subject have been thoroughly confirmed, not only by Theiler and other experimental workers, but also by the facts observed in practice. Heartwater has never been identified in any case of disease contracted naturally in any area free from bont ticks, although affected flocks and herds have very frequently been moved from Heartwater districts into areas in which most, or practically all, of the other known South African ticks were abundant.

In view of the great importance of the tick in the spreading of the disease, and in connection with the problem of its suppression, a résumé of the recorded facts relating to its habits and life-history may be given here.

Amblyomma hebræum, the " bont," " variegated," or " tortoise-shell " tick, is one of the largest South African ticks, and the adults are easily recognized, the male and unengorged female by the colours of the scutellum on the back (from which the popular name is derived), and the engorged female by the size attained, which considerably exceeds that of any South African tick other than *Hyalomma ægyptium*, the " bont - leg " tick. *Amblyomma hebræum* is a " dropping off " or " three host " tick.

The engorged female drops from the host, and after a period which varies from about two weeks in the summer to even more than three months in the winter, lays thousands of eggs. According to the temperature prevailing, the eggs may hatch in from ten weeks (in summer) to about ten months, the average time being four to six months, and the hexapod larvæ hatched out may shortly afterwards succeed in gaining a host; it has been shown, however, that they may live as long as seven months before feeding. Once on a host the larvæ attach and engorge with blood, and they drop off again in most cases in about five to seven days; periods varying from four to twenty days have been recorded. The engorged larvæ undergo the first moult on the ground, the time taken for this varying from about one up to four months, according to the season and temperature. The nymphæ which emerge are eight-legged, like the adults, but have not the scutellum with the mosaic of colours. They can exist without feeding for quite six months, and when a host is gained they remain on it for about four to eight days (periods up to twenty days have been noted), before dropping to the ground and undergoing the second moult, which may occur in from twenty-five to one hundred and sixty days. The adults which emerge can exist for a very long time, certainly over eight months, before feeding; the sexes mate about five days after being placed on a host.

The female remains on the host from ten to twenty days, but the males may remain for months, and probably mate with several females. Finally the engorged female drops off, and the cycle recommences.

Under the most favourable conditions the complete life cycle requires a period of about nine months, and under unfavourable conditions it may take over a year, and even up to two years.

A. hebræum is notable amongst South African ticks not only on account of the long period of time required for its complete life cycle, but also for its somewhat stringent requirements as regards natural conditions of climate and shelter. The bont tick requires more favourable conditions with respect to warmth and humidity than the other South African ticks, and it is not found in the high or middle veld, but only in the warm, low-lying, generally more humid, " bush-veld." The adults are chiefly found on ruminants, selecting as a rule areas where the skin is softer, such as under the elbows, in the perineal region, and on the udder; the larvæ and nymphæ are also generally found on ruminants, and attach anywhere.

The bont tick has been found on horses, cattle, sheep, goats, dogs, the " wild dog," antelopes, and the ostrich, and it may even attack man. Apart from its rôle in transmitting Heartwater, it may directly cause local troubles, such as suppurating sores on the udder, and suppurating lesions on the teats leading to constriction of the ducts and sloughing, and it may also cause lameness when attached near the feet in sheep and goats (such lameness is very common in many parts of South Africa from infestation with the bont - leg tick, *Hyalomma ægyptium*).

Lounsbury showed that when a female bont tick engorges on a sick animal, the Heartwater infection does not pass through the eggs, and the larvæ hatched out do not transmit the disease. The disease is conveyed by: (*a*) nymphæ which fed on virulent blood during the previous larval stage; (*b*) adults which fed on virulent blood during the previous nymphal stage; and (*c*) adults which as nymphæ fed on non-infected and insusceptible animals, but derived from larvæ fed on an infected animal. Apparently the infection acquired by larvæ persists to the adult stage only in a proportion of the ticks, but the fact of this possibility has some practical importance in connection with the prophylactic measures adopted. Lounsbury also proved that the infection is ordinarily transmitted within twenty-four hours of the tick's attachment, and that infected nymphæ and adults transmitted the disease whether they were fed soon after moulting or after a lapse of six months or longer. He failed in a number of attempts to infect ticks by placing them on recovered animals, and this agrees with the negative results of the inoculation of the blood of such animals, obtained by Theiler, Spreull, etc. Apparently the blood may cease to be infective as early as the twentieth day after inoculation; it generally fails to produce a reaction after the lapse of about four to five weeks.

The recovered animal is therefore not a " reservoir " for the virus.

Period of Incubating.—In sheep and goats the natural period of incubation, after tick infestation, is generally about fifteen days, but it varies from eleven to eighteen days, and even a period of twenty-three days has been recorded. In cattle the period is about twenty to twenty-five days. After the inoculation of virulent blood, the period varies from five to fifteen days, or even longer; the shorter periods are obtained with intravenous inoculation and the use of larger doses of infective material, but the virulence of the particular strain and the degree of susceptibility of the experimental subject affect the result also.

Symptoms. 1. **In Goats and Sheep.**—The first change is a rise of temperature, and this may continue until 106° F., or even 108°, is attained; the fever generally shows nocturnal remissions, particularly during the first four days, and ultimately it usually drops quite suddenly to normal or subnormal, generally shortly before death, but sometimes on the preceding day (or even longer in the less common subacute type of case). In the cases which recover the fever is usually not quite so high, nor so prolonged, but that is not always the case.

In stabled (experimental) animals the first visible symptoms may be dulness, prostration, and lack of appetite, but under natural conditions one will probably notice first a tendency to isolation from the rest of the flock when grazing, and to lag behind when the flock is driven. In mild cases there may be no symptoms other than a rise of temperature, and peracute cases occur in which the animals die so quickly that no symptoms develop (other than the rise of temperature); such animals are often found dead on the veld or in the kraal.

In other cases the animal shows dulness, disinclination to move, loss of appetite, cessation of rumination, and, when driven, quickened respiration and a tendency to lie down. Many animals show striking nervous symptoms, which may develop quite early, or only shortly before death; the animal may bleat repeatedly, and a very common symptom is a constant movement of the jaws as in mastication, while the tongue is repeatedly projected forwards and withdrawn. The animal in some cases is seen to lick the ground; the eyes may squint, and salivation may be observed, foamy saliva appearing about the lips. The animal may turn in circles, and finally drops to the ground, generally lying on the side; the head is generally extended forward and upwards, and at times there may be marked opisthotonos. As the attack progresses, there may be localized muscular tremors, or more widespread and violent muscular spasms, and Spreull records a characteristic "galloping" action of the legs.

The nervous symptoms may persist with little change for some considerable time, or they may occur in fits which commence and end quite suddenly; in the latter case an attack may often be excited by any interference or stimulation, and interference during an attack will generally increase the severity of the manifestations. Either constipation or diarrhœa may occur, but in the majority of cases the bowels act normally; during a nervous attack with muscular spasms, however, normal fæces and urine may be repeatedly passed in small quantities. Death usually follows fairly rapidly, especially if more than one nervous attack has occurred, and it is quite exceptional for an animal to recover after having shown the nervous symptoms at all clearly. Death may be preceded by general convulsions, but in other cases the animal passes into a state of coma.

The disease generally runs its course in from two to six days after the first rise of temperature, but in some cases it is considerably prolonged.

2. **In Cattle.**—The symptoms shown by cattle are very similar indeed to those noted in goats and sheep, and they were well described by Theiler in a study of both natural and experimental cases, published in his *Annual Report* for 1903–4.

As in small stock, there are mild cases in which a temperature reaction may be noted, but in which ordinary clinical symptoms are absent or quite insignificant. Other cases show dulness, prostration, impairment or loss of appetite, cessation of rumination, and other general symptoms. Many cases, however, show striking nervous symptoms, including the peculiar movements of the jaws and tongue already noted in connection with sheep and goats. Other symptoms which may be observed are muscular tremors, nodding of the head, a peculiar blinking of the eyelids, and a glaring eye with injection of the sclerotic vessels. The animal may wander in circles, or if tied to a post may repeatedly encircle it, and it may push with the head into objects; finally it goes down. There may be localized or more or less general muscular tremors, and in the later stages stronger muscular spasms; the legs may move with an action as if trotting. Attacks may be initiated or markedly increased by even slight stimuli, such as a light and touch. In some instances the animal bellows repeatedly, and Theiler records cases of biting of the ground, and even of repeatedly biting the leg on which the head was resting. Finally coma supervenes.

The duration of the disease from the first appearance of fever is usually about six days, but the period of visible illness in fatal cases is generally only a couple of days, or less.

As in small stock, peracute cases occur in which death supervenes so rapidly that no clinical symptoms are noticed; under natural conditions such animals are quite unexpectedly found dead in the veld.

Mortality.—This varies considerably, and it is impossible to give any precise figures which would be applicable to all cases. In the first place, the different species attacked show variations in susceptibility according to breed and age, as well as considerable individual variations in resistance, and a greater degree of resistance is likely to be found amongst animals which have been bred in or near Heartwater areas. Moreover, there are, without doubt, strains of varying degrees of virulence naturally occurring in different localities, and in all cases the mortality (as well as the incidence) is likely to be heavier in summer than in other seasons.

In goats and sheep many experiments have resulted in a mortality of 100 per cent, but under natural conditions the mortality is usually very considerably lower, and about 60 per cent would be a more probable figure; Spreull gives figures of 50 per cent for merino sheep and 80 per cent for Angora goats.

With respect to cattle, the published experimental data show wide variations, most probably due partly to the use of different strains, and partly to the accidental selection in some cases of animals already possessing a considerable degree of resistance. Thus Lounsbury only obtained two deaths after inoculating five calves, and no deaths at all in three adult cattle, whereas Theiler, working in the Transvaal, killed six out of seven inoculated animals ranging from eighteen months to two years in age, and later, after inoculating four three-year-old oxen, he observed fatal reactions in three of them. Theiler's results, and the facts observed in practice, particularly in the Transvaal low veld, show that Heartwater *may* be a very fatal disease for cattle, and newly imported stock show the greatest susceptibility.

Lesions observed post mortem.—On reflecting the skin it is often observed that the vessels are deeply injected; some subcutaneous œdema may occur. Rigor mortis and coagulation of the blood usually take place normally. The characteristic " Heartwater " is generally seen in sheep and goats, especially in natural cases (but it is very frequently absent in cattle). The amount of liquid in the pericardial cavity may show only a slight increase over the normal, or the pericardium may be greatly distended with liquid. In a perfectly fresh carcase the liquid is practically always clear and light yellow in colour, but if the autopsy is made later it may show a reddish tinge, and exceptionally it may have coagulated in the unopened pericardial sac. In any case the liquid generally coagulates well after the pericardium is opened, or if it is collected in a vessel. Similar exudates may or may not occur in the other serous cavities, particularly the pleural cavity. A case with a good " Heartwater " lesion usually shows some pleural and peritoneal exudate, but this is not necessarily the case.

The lungs may appear normal, but in acute cases generally show some degree of œdema, with the presence of froth in the bronchi, and even in the trachea. The epicardium may show petechiæ, and the endocardium, especially in the left ventricle, often shows numerous petechiæ, and even extensive ecchymoses. The spleen is generally only slightly enlarged, with pulp of softer consistence than normal. The liver may appear to be normal, but is usually congested, and the kidneys also sometimes show congestion. The bladder and urine show no obvious change.

The abomasum may show little, but there is often some degree of gastritis; the mucous membrane may be thickened and œdematous, and it may show diffuse hyperæmia, numerous scattered petechiæ, or exceptionally hæmorrhagic patches and streaks. The small intestine may also show little or nothing abnormal to the naked eye, but it usually shows some inflammatory changes which may affect the whole length of the bowel or only certain parts. The mucosa is generally thickened, and may exhibit changes varying from a diffuse reddening or mere injection of vascular " arborizations," to the presence of collections of small petechiæ, or less commonly, the occurrence of well-marked patches or streaks of congestion, or even hæmorrhagic patches. Such changes, when present, are generally better marked in the ileum, in its posterior portion, and in this situation Theiler has even seen necrosis of the mucous membrane. Peyer's patches show no abnormality. In the large intestine one may find changes similar to those seen in the small bowel, but they are often absent.

Some differences are to be seen between the naked-eye lesions observed in cattle and those which occur in sheep and goats. The typical " Heartwater " is generally observed in natural cases in sheep and goats, although it is not quite constant. It is less constantly found in sheep or goats inoculated with virulent bovine blood, and it is quite frequently absent in cattle, although in a minority of cases it constitutes a striking feature. The gastro-enteritis tends to be more severe in cattle, and Theiler has remarked that in certain cases the enteritis seen may even recall the lesions occurring in Rinderpest, apart from the fact that Peyer's patches are not involved in the former case.

No characteristic microscopic lesions have been described; the blood in an uncomplicated case shows no abnormality.

Diagnosis.—(*a*) In all cases one is naturally guided to some extent by considerations of locality and season, and by the history of the outbreak. Thus one could safely eliminate Heartwater in connection with any outbreak occurring on the high veld, unless the history showed that the affected animals had been brought from low bush-veld within the usual incubation period of that disease.

(*b*) The clinical symptoms may be almost diagnostic, when the nervous symptoms are well developed, and the association of such symptoms with a high temperature is of some significance, although individual cases might be seen only in the later stages, when the temperature had fallen to normal or sub-normal. On the other hand, in many cases no positive diagnosis could be made on clinical grounds, particularly in those cases in which death occurs before any symptoms have been noted.

Apart from the nervous symptoms, which may not be present or at all well marked, the symptoms shown are common to a number of febrile diseases which are quite likely to occur in areas liable to Heartwater, and no safe diagnosis can be based on their presence.

Certain other specific febrile diseases occurring in South Africa are apt to give rise to nervous symptoms in cattle ; for instance some excitement, bellowing, and an inclination to charge any human being or animal approaching, may be seen in an acute case of Redwater, but these symptoms are not quite the same as those seen in a typical Heartwater case. Similar symptoms of excitement are seen in some cases (loosely termed " Gall Sickness ") in which the temperature is little, if at all raised, and in which all the well-known specific diseases can be excluded ; such cases are presumed to be due to the ingestion of some poisonous plant, and, as in acute Redwater, the *localized* nervous symptoms seen in Heartwater (such as the peculiar movements of jaws, tongue, and eyelids) are not shown. There are certain outbreaks, however, which are also believed to be due to some form of vegetable poisoning, and in which nervous symptoms more closely resembling those of Heartwater are prominent, and most of the nervous manifestations mentioned might be exhibited in some cases of Snake-bite (these would, however, be only sporadic cases).

In most of these cases there are features which would at least help to differentiate them from Heartwater cases, and this applies particularly to the temperature.

(*c*) The post-mortem lesions may also be very typical, and if combined with a characteristic clinical history, will then leave very little room for doubt. In sheep and goats a really typical post-mortem picture is not likely to be found in any known condition other than Heartwater, but in atypical cases the lesions present are very similar to those which may be encountered in a variety of other conditions, and particularly in conditions of as yet undetermined origin, believed in many cases to be associated with the ingestion of poisonous plants.

A specific tick-transmitted Gastro-enteritis of sheep described by Montgomery in East Africa should also be remembered in this connection.

There should, of course, be no difficulty in differentiating cases of debility due to parasitic invasion, with poor condition, anæmia, the absence of acute lesions, and the presence in the serous cavities of thin, usually incoagulable transudates.

In cattle the post-mortem lesions found are very frequently atypical, and most or all of the lesions are similar to those encountered in other more or less common conditions. Thus similar lesions may be found in acute cases of Redwater and in East Coast Fever, although these conditions are likely to show other lesions not found in Heartwater (such as the usually greatly enlarged spleen in Redwater), and can, of course, be differentiated microscopically. Such lesions are also found in some peracute cases of Lamziekte, and in cases which have at present to be ascribed to vegetable poisoning.

(*d*) No microscopic diagnosis can be made. As previously stated, Heartwater produces no recognizable blood changes, but it should be remarked that most of the cattle which contract the disease are already immune to several other South African diseases, and harbour various protozoa which are liable to multiply during Heartwater reactions, and therefore to be detected in blood smears. This is particularly the case with *P mutans*, which can very frequently be found in practice, and *P. bigeminum*, *Anaplasma marginale*, *Spirochæta theileri*, and *Trypanosoma theileri* have all been noted in similar cases (see Theiler, *Annual Report*, *G.V.B. Transvaal*, 1908–9, page 59).

(*e*) In all cases of doubt, when it is considered important to ensure an accurate diagnosis, recourse may be had to the inoculation, with the blood of sick animals, of goats or sheep, kept under close observation, and under conditions excluding the possibility of the accidental contraction of Heartwater, or of any probable disease liable to be confused with it. As previously stated, goats or sheep inoculated with virulent bovine blood *may* fail to show the most characteristic lesions, but if the virus is passed from sheep to sheep (or goat) for a few generations, the typical picture is soon obtained.

Those particularly interested in this question should refer to the account given by Theiler (*Annual Report*, 1903–4) of the inoculations and sub-inoculations made by him in order to prove that a certain Ox 448 suffered from naturally contracted Heartwater. The example in question shows clearly how a number of inoculations may have to be made in order to overcome the difficulties arising from the production of further atypical reactions and lesions in susceptible animals, and from the accidental selection as experimental subjects of animals possessing a considerable degree of resistance.

Preventive Measures.—The most important of the prophylactic measures needed are naturally those directed towards the eradication of the bont tick, and of all such measures constant short-interval dipping of cattle is the most successful. Reference to the facts established in connection with the life-history of the bont tick will show that to be sure of catching every tick that may infest the animals at any time, the cattle should be dipped at least every four days. In practice *five-day dipping* will catch the great majority of the ticks which are picked up, and this interval is now the one most commonly adopted in Natal and the Transvaal (where the dipping is primarily directed against the ticks which transmit East Coast Fever).

Fortnightly dipping will naturally kill many ticks, and will ultimately reduce their numbers to insignificant proportions, but five-day dipping is much to be preferred, and is far more certain and very much more rapid in its effect.

The dipping fluid should have sodium arsenite as its basis, and the " Laboratory dip " of Watkins-Pitchford, or a solution of arsenite of soda of corresponding strength, is to be recommended, the strength varying according to the dipping interval chosen (see "East Coast Fever").

Such dipping has proved very successful in many areas in reducing the number of ticks of all species, and in reducing the incidence of Heartwater to quite insignificant proportions.

In a badly infested bush-veld area, however, it cannot be expected that complete eradication of the bont tick will be attained within a few months or a year. The comparatively very long periods during which the tick in its various stages may live before gaining a host, and the long periods necessary for the laying and hatching of the eggs, render it possible for the eradication to be long delayed. Another important fact is that an infected larva may, as a nymph, feed on some insusceptible (and undipped) host, such as a dog or wild antelope, and transmit the infection in its adult stage. In view of the long periods which may be passed in the various stages before the adult is developed, and of the long survival period of the adult, it is obvious that some cases of Heartwater would still be liable to crop up even eighteen months or more after the institution of regular five-day dipping of all cattle, and the presence on the same veld of any susceptible goats and sheep (which, of course, are not subjected to short-interval dipping) would naturally extend the period during which such cases were liable to occur.

The above-mentioned facts do not merely indicate theoretical possibilities, but explain what has actually been found to occur in a number of instances in recent years, during which many low-veld areas, previously unsettled, have been laid out in farms or taken up in larger blocks for ranching.

Another prophylactic measure is the widespread and old-established practice of *grass-burning* ; from some points of view this practice is quite harmful, but under certain conditions it appears to be necessary at the present time, and without doubt many ticks may be destroyed in this way, the number depending largely on the season. Only those ticks which are actually on the grass are likely to be destroyed, and during the winter and early spring comparatively few ticks are to be found there. The later in the season the burning is done, the more tick-destruction is likely to be effected, but in most cases the farmer is guided, in his selection of the season, more by considerations relating to the provision of fresh grazing.

It would also be possible to starve out the ticks, provided that a limited and enclosed piece of veld could be kept entirely free from any of the hosts of the bont tick, but it would not be easy in practice to exclude all possible hosts for a sufficient length of time (a minimum of fourteen to fifteen months).

Such methods of tick eradication as ploughing and cultivation, and dressing the land with various substances, have obviously an extremely limited application in the areas likely to be affected with Heartwater.

The oldest and simplest method of checking an outbreak is to move the animals, and when it is possible to move the flock or herd at once to higher ground, where the bont tick cannot multiply or thrive, no further cases are contracted. Where it is impossible to move to high veld, and it is desired to move on to ground believed to be more or less free from Heartwater, although infested with the bont tick or liable to become so, recourse may be had to the " quarantine camp " method largely used in connection with East Coast Fever. In this case two successive moves would be required, the animals being kept for three to four weeks in each of the two places, and all affected animals picked out and returned to the original ground (where the rapid termination did not render this unnecessary).

No certain method of *artificial immunization* has yet been worked out and applied on a large scale, and the success which has been obtained with dipping has rendered such a process less urgently required than was formerly the case, although it would be by no means unwelcome or unnecessary. Spreull obtained some success in a number of experiments in which the blood of recovering cases was used as a vaccine, although he found the immunity produced in many cases to be insufficient to protect against natural infection ; later he injected small doses of virulent blood subcutaneously.

Theiler showed that it was possible, by hyperimmunization of recovered cattle, sheep, and goats with virulent blood, to produce a protective serum, but he did not continue the experiments to the point of determining all the details necessary for an exact process of immunization suitable for application in the field. His work indicates that such a method is quite feasible, but our inability at present to preserve the virus *in vitro* for longer than forty-eight hours is a practical difficulty, and one would have to take into consideration the natural occurrence of strains with varying degrees of virulence.

Curative Treatment.—No drug has been found to give any promising or useful results with this disease. In cases which are not too acute, good nursing will increase the chance of recovery, and such measures as isolation, placing the animal in a cool shady spot, and the provision of green food are indicated. W. H. A.

BLUETONGUE IN SHEEP

Bluetongue is a specific febrile disease of sheep, caused by an ultravisible virus, and characterized by catarrhal inflammation of the mucous membranes of the mouth, nose, and intestine, very often accompanied by inflammation of the laminæ of the feet.

The name " Bluetongue," a literal translation of the Dutch " Blaauutong," was given to the disease by the farmer on account of the bluish colour frequently shown by the tongue, and by the buccal mucous membrane generally. The disease is also sometimes termed " Bekziekte " (Mouth-sickness) and " Fever," and in veterinary literature has been referred to as " Malarial Catarrhal Fever " (Hutcheon) and " Catarrhal Fever."

The disease appears to have occurred in South Africa for a very long time, and in Cape Colony it soon attracted the attention of Hutcheon, who gave a good description of the symptoms, and of the conditions under which outbreaks were recorded.

During the first few years of this century it was the subject of investigations carried out by Dixon, Spreull, and Robertson in Cape Colony, by Theiler in the Transvaal, and in Natal by Watkins-Pitchford. These investigations established a number of important facts relating to the nature of the disease, and led to the introduction by Theiler of a very successful method of vaccination which is now applied on a very large scale.

Geographical Distribution.—The disease has been recorded in practically all parts of the Union of South Africa, and in Rhodesia, the Bechuanaland Protectorate, the territories formerly known as German South-West Africa and German East Africa, and in British East Africa. It is probable that the disease has a considerably wider distribution in Africa than is indicated above, and that, like Horse-sickness, it occurs also in the northern part of the continent.

In South Africa the disease is found in high, middle, and low veld, but it is more commonly and constantly observed in the areas subject to a relatively heavy rainfall.

Species affected.—No natural outbreak has ever been recorded in any animals other than sheep. In practice the worst outbreaks appear to occur amongst Merinos, Merino crosses being somewhat less affected, while Africander and Persian sheep are said to suffer much less, but under experimental conditions Theiler found little or no difference in susceptibility between the various breeds. Sheep of any of these breeds show fairly wide individual variations in susceptibility, and the considerable degree of resistance which may be encountered is probably "natural" in some cases, and in other cases acquired as a result of previous, and possibly very mild, attacks. Young animals are, as a rule, more susceptible than adults.

Spreull found that inoculated goats did not develop any visible symptoms, and as a rule showed no temperature reaction, but that their blood was infective for sheep from about the fifth to the tenth day after inoculation, and sometimes for a longer period ; he was able to infect sheep after passing the virus through a series of ten goats. Similarly the blood of inoculated calves was found to be infective, although the calves showed no signs of a reaction. Theiler failed to infect the horse, mule, or ox by inoculation, and Robertson obtained negative results with the horse and dog.

Cause.—The disease is caused by an ultravisible organism or " virus " which is present particularly in the blood, and which has been shown by Theiler (confirmed by Robertson) to pass through the Berkefeld filter.

It has not yet been cultivated *in vitro*, but it can be preserved for a long time ; the virus is not destroyed by the putrefaction of the infective blood containing it, as was demonstrated by Theiler, nor by desiccation of virulent blood, although it suffers a gradual attenuation. When to defibrinated virulent blood one adds an equal volume of a 50 per cent solution of glycerine in water, containing also 0·25 per cent carbolic acid, the mixture generally retains its virulence more or less unaltered for a year, or even longer, but in time it will show attenuation.

Transmission of the Disease. 1. *Experimental transmission.* — Bluetongue is easily communicated artificially by the inoculation of infective material under the skin, into a vein or into one of the serous cavities; Theiler and Dixon failed to communicate the disease by drenching, although Watkins-Pitchford claimed to have succeeded in this way. Virulent blood is most commonly used, defibrinated or citrated, but plasma and serum are also infective, and the disease has been set up by the inoculation of spleen pulp and of various exudates. The blood is infective during the reaction, and for about a month after it; exceptionally it may remain virulent for a considerably longer period, and in one instance Spreull conveyed the disease with blood taken fifty days after the temperature had fallen.

In all cases, some time after the animal recovers, the blood loses completely its infectivity as tested by inoculation, and the recovered animal is therefore presumed not to act as a " reservoir " for the virus, although it is theoretically possible that the virus may be present in a form not directly inoculable to sheep, and requiring first to undergo changes in the body of some intermediate host.

The disease has been induced by the subcutaneous inoculation of only one drop of virulent blood, and doses varying from one to twenty cubic centimetres produce the disease equally promptly and equally severely, but still larger quantities, such as 50 c.c., are likely to produce severe and fatal reactions (Theiler).

There are great individual variations in susceptibility, and blood taken from a mild case may induce very severe reactions in less resistant subjects.

2. *Natural transmission.*—Bluetongue occurs under conditions practically identical with those which are associated with the occurrence of Horse-sickness, and there is reason to suppose that the two diseases have a similar mode of natural transmission. It has even been suggested that the two diseases are identical, although this can hardly be maintained in view of the experimental evidence to the contrary brought forward by Theiler and other workers. Bluetongue may be said to occur in all known Horse-sickness areas, as far as South Africa is concerned, but the converse probably does not hold good, Bluetongue having a wider distribution.

Hutcheon long ago suggested that transmission is effected by a biting insect; and although direct and definite proof is still lacking, the hypothesis that the disease is carried by a nocturnal flying insect (very probably a mosquito) is almost generally accepted at the present time. The evidence in favour of this view may be summarized thus:

(*a*) The incidence of the disease varies greatly from year to year, and a bad Bluetongue year is almost, if not quite, invariably a year of heavy summer rains.

(*b*) Bluetongue is ordinarily a disease of the late summer or autumn; the season in the Transvaal includes most commonly the months of March and April, and sometimes May also, but early rains may be associated with the appearance of the disease early in the summer (even December or earlier).

(*c*) All observers in the field are agreed that the disease is stopped by the first good frost of autumn or winter, although a very slight frost may not be effective. A few further cases are liable to develop symptoms after the frost, as some of the sheep may have been infected very shortly before its occurrence.

(*d*) The disease occurs on high, middle, and low veld, but relative altitude has a marked influence, and although the veld at the foot of a hill may be highly dangerous, sheep kept well up on the side or on the summit of the hill may be quite free from the disease.

(*e*) The neighbourhood of water is always more dangerous; this applies particularly to swamps or " vleis " and ponds or " pans," but also to dams, streams, and rivers.

(*f*) The disease is contracted at night, and sheep can be protected by keeping them during the dangerous nocturnal hours in a shed or on high ground.

(*g*) Dipping has been considered to exercise a protective influence, and shorn sheep are said to contract the disease more easily than those with long wool.

(*h*) The disease is easily inoculable, but not directly contagious or infectious.

(*i*) Watkins-Pitchford kept sheep in gauze-covered pens on a river bank at night, during the Bluetongue season, and they remained healthy, although control sheep, tied up at night outside the pens, contracted the disease.

Period of Incubation.—The period of incubation under natural conditions has not been accurately determined, but after inoculation it averages four days, and rarely is as short as two days.

Symptoms.—The course of the disease may be acute or subacute, and abortive cases are by no means rare, especially in smaller and milder outbreaks.

The symptoms may be divided into three groups, viz.: general symptoms, local manifestations in the region of the mouth, and those about the feet.

(*a*) *General.*—The first sign of the disease to be detected under experimental conditions is usually a raised temperature, although in a number of otherwise typical cases little or no pyrexia may be recorded throughout the attack. The temperature may rise either gradually or quite suddenly, and may reach 106° F. or even 107°; as a rule the temperature curve is irregular, with well-marked remissions. The fever reaction lasts on an average for about six days, but extremes of two and eleven days have been recorded in experimental cases (Theiler). Some signs of general malaise may be shown a couple of days after the temperature rises. In a proportion of cases no further symptoms are to be detected, and the animal recovers without serious loss of condition; this abortive form of the disease is also the rule after vaccination.

In other cases, however, after a few days the mouth lesions develop; the animal is then depressed, the appetite may be lost or the animal may be unable to feed although willing to do so, rumination may be suspended, condition is lost very rapidly, and in acute cases the patient goes down and is unwilling to rise or move; the head may be turned to the flank. Still later the development of the lesions in the feet may cause lameness, pain, reluctance to move, and the animal will probably again go down and refuse to rise. Diarrhœa appears in some acute cases, and is a very unfavourable sign, such cases generally soon collapsing, but as a rule the fæces are firm and coated with an excess of mucus. Spreull records the occurrence of great thirst, and states that if sheep have free access to water at the time when they are beginning to feed again, they may vomit, with the possible sequel of pneumonia set up by inhalation of ingesta, but Theiler saw vomiting only in one case. The appetite may be completely lost during the acute stage, but it may persist throughout the attack, in spite of the development of mouth lesions which may render feeding very slow and difficult, or even impossible, and although rumination may be suspended, as usually happens in a bad case during the acute stage.

(*b*) *Local manifestations in the region of the mouth.*—Changes in the mouth usually begin to become visible two or three days after the temperature rises, although they may not attract attention until a little later. At first there is a diffuse reddening of the buccal mucous membrane, although in very acute cases it may show a bluish tinge at an early stage, and shortly afterwards the animal begins to exhibit occasionally a movement of the tongue suggestive of licking the lips.

In some abortive cases even these mild symptoms are lacking, and in others resolution occurs after the diffuse reddening has persisted for a few days.

In many cases, however, further symptoms soon appear; swelling of the lips and nostrils may become noticeable; there may be frothy saliva on the lips, a serous discharge from the nostrils, and less frequently from the eyes. As the disease progresses the nasal discharge becomes catarrhal in character, and later may show some intermixture of blood, and the swelling of lips and nostrils increases. The buccal mucous membrane has now a bluish or purple colour (the feature to which the disease owes its name), but in some situations more pronounced local œdema may cause pallor, particularly on the incisor pad and, in places, on the gums. More or less extensive shedding of proliferated epithelium may now commence on the lips, and is generally first noticeable at the junction of the mucosa with the skin.

In the majority of the cases seen in outbreaks of ordinary severity, recovery begins after the disease has shown the above-described symptoms for a few days.

In more severe cases, however, in various situations one may observe sloughing of epithelium, with the result that raw sores, which may show some adherent blood-clots, are formed. These are found particularly inside the lips (especially the upper lip), on the dental pad and gums, and on the tongue, towards its tip, less commonly on the cheeks and hard palate. The nasal discharge is now thicker and more profuse, there is increased swelling of the lips and about the nostrils, and similar sores may be seen outside the mouth, between the nostril and the upper lip, on the lips themselves, and even in the nostrils. The sloughing of epithelium on the tongue may be very extensive, although the base is not affected, and the organ may be considerably swollen.

Death may occur in this stage, especially when diarrhœa appears, but as a rule the mouth lesions retrogress after about four or five days.

(*c*) *Symptoms associated with foot lesions.*—As a rule these, when present, begin to appear at the time when the mouth lesions are starting to recover. In many cases lesions of the feet do not develop at all, or they are so slight as to attract no attention, but in some outbreaks the majority of the affected sheep may show considerably more severe lesions of the feet than are shown in other, and not necessarily less serious, outbreaks. The severity of the foot lesions is not necessarily proportional to that of the mouth lesions, nor to that of the general symptoms, although it need hardly be stated that the foot lesions are likely to be very poorly developed, if seen at all, in mild outbreaks of the disease.

One probably notices first that animals which were beginning to recover from the acute stage, and with the healing of the mouth lesions were commencing to feed better, are again disinclined to move, while the appetite may again be partially or completely lost. If the affected animals are forced to walk, they may be lame, or may show a stiff gait and arched back, and the gait may be similar to that seen in a typical case of laminitis in the horse. A little later the animals may go down and show no inclination to move, or they may be seen painfully crawling about on the knees to feed. The feet are hot to the touch and tender to pressure, especially at the heels, and if the hoof wall is rather thin and transparent (as in lambs), the whole foot may appear abnormally red, perhaps with a bluish tinge, which is more pronounced later. The coronet is somewhat swollen and tender, and shows a reddish or blue line or band ; in an uncomplicated case there are no lesions of the skin of the extremity, other than some degree of swelling with perhaps hyperæsthesia towards the coronet. These changes may involve all four feet, or only two feet, and in the latter case the two fore feet are most commonly attacked ; occasionally only one foot is affected.

As a rule these symptoms abate after a few days, but in some severe outbreaks in the field shedding of hoofs may occur in a number of cases. In the majority of cases both the mouth and the foot lesions heal and disappear, but the animal, after an attack of any severity, is left in an emaciated and weak condition. If the debility be excessive, the animal may lie persistently, make little effort to feed, and die from exhaustion and inanition, but in most cases, especially if well tended, the debilitated animals slowly recover condition.

In some outbreaks loss of wool occurs as a not uncommon sequel ; this process may be partial and unequal, leaving a number of bare patches, or it may sometimes be very extensive, involving almost the entire fleece. The condition is sometimes known as Vuurziekte (" Fire-sickness ") from the reddish colour of the exposed skin, but apart from its colour and a superficial roughening the skin shows no other abnormality, and no signs of pruritus are seen. Eventually the exposed skin is covered by the growth of normal wool.

Mortality.—The mortality varies within fairly wide limits, and it appears that strains of varying degrees of virulence occur in nature. In some districts outbreaks are generally attended by a much heavier case-mortality than that recorded in some other districts, and generally speaking the heaviest case-mortality is seen in lower-lying and well-watered areas, where the disease is apt to appear every season. With respect to Bluetongue areas in general, during dry seasons few outbreaks are likely to be recorded, and as a rule such outbreaks as do occur are likely to be characterized by milder reactions and a low mortality. The cases which are observed in the normally drier parts of the country (such as many parts of Bechuanaland and of the western districts of the Transvaal), where for several seasons very few cases may occur, or even none at all, tend to be of the same milder type, except in occasional, and generally exceptionally wet, seasons, when the extensive outbreaks which may then occur are also apt to be notable for the much greater severity of the cases The extent to which the affected animals are cared for may also naturally affect the case-mortality.

In general Bluetongue cannot be regarded as a very fatal disease ; in Theiler's work the experimental cases gave a mortality of barely 20 per cent. In natural outbreaks the mortality may be very low, from 3 to 5 per cent succumbing, and in fact where only a few animals are affected none may die, but in other places, or at the same places in other seasons, 10 to 30 per cent, or even as many as 60 per cent, of the sheep in the affected flocks may be lost. In the majority of outbreaks the general loss in condition of the flock, with possibly very serious consequences in the following winter, and the damage to the wool, cause more concern to the farmer than the actual deaths directly attributable to the disease.

Post-mortem Lesions.—When the animal dies during the acute stage of the disease, the mouth will show characteristic lesions, and if the tongue is extensively involved it is of some importance to note that towards the pharynx it is not affected. The mucosa of the septum nasi is usually much congested.

If death has occurred at a later stage of the disease, one may still be able to see some signs of the mouth lesions, although the more typical appearance has given place to that of one or two irregular raw-looking ulcers, and at this later stage some abnormality may be detected in the feet.

The pharynx and œsophagus are generally normal ; the lungs are also usually normal, or only slightly œdematous, but in an acute case they may show pronounced œdema, with the presence of much froth in the bronchi and trachea. An increased amount of liquid may be found in the pericardial cavity, and this may also be seen in the pleural, and less commonly in the peritoneal, cavities. The epicardium may show petechiæ, and some petechiæ are nearly always found under the endocardium, especially in the left ventricle, where even large ecchymoses may be seen. The blood is usually well coagulated.

The liver as a rule is congested, and the gall-bladder is usually filled with normal bile. The kidneys may show congestion in the cortex, but often appear to be œdematous; the capsule strips easily. Bladder and urine normal. The spleen is usually moderately enlarged, with softened pulp.

In a very acute case the rumen may show reddish patches and streaks, and also reddening of the top of the papillæ and pillars. The reticulum may show similar lesions, but the omasum is only rarely affected; its contents are generally quite soft.

The abomasum is usually affected, but shows various degrees of change ranging from a slight patchy or uniform congestion, or the presence of scattered petechiæ, to conditions in which the mucosa is severely affected. It may be considerably swollen, a change which is more easily appreciated in the folds, and also very congested, being even purplish in colour. The lesions may affect the whole organ, only one end of it, or discrete patches.

The small intestine is generally involved, and the lesions are as a rule well developed in those cases which were associated with diarrhœa; the bowel may be affected throughout its whole length, or only in larger or smaller patches. The lesion appears as a thickening and congestion of the mucosa, which is covered with mucus; Peyer's patches are often congested.

The large intestine may be inflamed throughout its whole length, but it usually shows only some patches of congestion. The bronchial and mesenteric lymphatic glands are generally somewhat enlarged and œdematous, and may appear congested.

In the more commonly observed cases, in which death occurs from exhaustion after the acute attack is over, the mouth lesions may probably have disappeared entirely, and the feet may show no obvious abnormality, while most of the above-mentioned lesions may be absent. In such cases one only sees emaciation, abnormal pallor of the muscular and other tissues, an increased amount of liquid in the serous cavities, etc.

No characteristic microscopic lesions have been described, and it is not possible by ordinary microscopic examination to detect any abnormality in the blood.

Diagnosis.—(*a*) The locality, season, and history of the outbreak should receive due consideration.

(*b*) The clinical symptoms are very often quite sufficient to establish the diagnosis, and especially when a number of cases can be seen, as in some the mouth lesions are almost certain to be typically developed, and, if there are cases in different stages, foot lesions may also come under observation simultaneously. Even when other external signs are lacking, the peculiar "licking" movements of the tongue may attract attention to the mouth. The association of such lesions with a high temperature and a rapid loss of condition is significant. It may be very difficult or quite impossible to diagnose particular cases clinically, where the mouth lesions are not well developed, or not well shown at the time when the animal is examined.

(*c*) The post-mortem lesions are usually fairly typical if the animal died during the acute stage of the disease, but are of little or no diagnostic value in cases of long duration. Apart from the lesions in or about the mouth and nostrils, most of the other lesions seen in acute cases are also met with in connection with other fairly common pathological conditions in sheep; for instance, very similar appearances may be seen in atypical cases of heartwater, and in various cases (generally known as "Gall-sickness") which are believed to be attributable to some forms of vegetable poisoning.

(*d*) No microscopic methods of diagnosis are at present available.

(*e*) Although in practice it is not usually necessary, it is always possible to control a diagnosis by experimental inoculation, and the fact that blood from a sick animal can so easily be preserved for a long time without loss of virulence renders it practicable in all important cases for the tests to be carried out in a central institution (however distant) or other place where natural infection can more certainly be excluded.

Differential Diagnosis.—In connection with the diagnosis of Bluetongue, the following diseases may be briefly considered:

1. *Foot and Mouth Disease* would, of course, tend to spread far more rapidly, and in addition to attack animals other than sheep, particularly cattle. Moreover the predominance of the foot lesions, and the nature of the mouth lesions if present, are distinguishing features.

2. *Sheep Pox* should easily be differentiated in a typical outbreak, as the external lesions differ considerably both in their nature and distribution, although in any outbreak some individuals may fail to develop the characteristic signs. Outbreaks have been recorded, however, both in European countries and in African territories subject to Bluetongue, in which few or no animals have shown the appearances considered typical of Sheep Pox, and some difficulty was experienced in differentiating between Bluetongue and an atypical form of Sheep Pox which occurred in German South-West Africa. In this form of Sheep Pox no typical variolous lesions were found anywhere on the skin, and in many cases at least it was rather difficult to find cutaneous lesions at all, but they were generally to be found particularly

on the inferior aspect of the tail. These lesions generally took the form of hard nodular swellings in the skin which did not develop into vesicles or pustules.

Montgomery also recorded a somewhat atypical form of Sheep Pox occurring in British East Africa.

In all cases where the diagnosis could not be settled definitely by the aspect and distribution of the lesions, it would be possible to arrive at a decision by the inoculation of blood, which is not infective in Sheep Pox, and of material from the local lesions.

3. A number of pathological conditions occurring in sheep and associated with mouth lesions have been described under various names such as Malignant Aphtha, Aphthous Stomatitis, Orf, and Contagious Pustular Dermatitis. Some of these conditions present a number of features in common with Bluetongue, and perhaps particularly those conditions which are associated with foot lesions, but in most cases there are well-defined points of difference. Thus in many cases the lesions affect particularly the muzzle, and the interior of the oral cavity may show practically no change, while in many instances the lesions of the extremities are by no means confined to the feet and coronets, but occur much higher on the limbs also, and in other situations such as the udder. In most cases close observation of the lesions in various stages would reveal specific differences in their nature and mode of development, *e.g.* the presence of discrete papules, vesicles, etc. In 1917 the writer saw a widespread outbreak of this nature, in Bechuanaland and the Western Transvaal, affecting sheep of all ages and both sexes, and associated with few, if any, deaths. The lesions, which were accompanied by a fever reaction, were to be seen chiefly on the skin surrounding the mouth (both upper and lower jaws) and nostrils, and took the form of hard papules which soon became pustules, and tended to become confluent in the pustular stage. There was much inflammatory swelling of the whole muzzle, and in a few days there was extensive desquamation, leading to the formation of thick dry crusts. Healing was generally rapid, and in most cases, when the animals were well tended, the loss in condition was noticeable but not excessive.

The condition would appear to resemble rather the " Contagious Pustular Dermatitis " described by Berry in 1901 (*Journ. Comp. Path. and Therapeutics*, vol. xiv. p. 307) except that, in all the cases seen, the lesions were confined to the muzzle. The disease appeared to spread rapidly from farm to farm in a manner suggestive of direct infection rather than transmission by an intermediate host, and although some farmers mistook the condition for Bluetongue, there were many who clearly distinguished it on account of the appearance and situation of the lesions (chiefly or entirely *outside* the mouth).

4. *Geeldikkop* is easily distinguished by the swelling of the upper part of the head also and particularly of the ears, and in a later stage by the tendency to necrosis. There is also jaundice, and, at least in most cases, there are no foot lesions, and no lesions inside the mouth resembling those of Bluetongue.

Prevention.—Where circumstances permit, the disease can be more or less entirely avoided by keeping the sheep at night on high ground, or in a shed. Frequent dipping of the sheep, or smearing them with substances of such a nature as to repel insects without injuring the sheep or their wool, is hardly practicable, and other measures which would tend to lessen or prevent the incidence of the disease, such as drainage of swamps, are hardly of practical importance in most areas liable to outbreaks of Bluetongue.

The most practical means of prevention is therefore the production of an active immunity. The first successful method of immunization was a simultaneous serum-virus method worked out by Spreull, and the results obtained were good, but the nature of the method rendered it somewhat costly. Watkins-Pitchford produced a vaccine by attenuating virus by the action of heat, and the method is reported to have given satisfaction when put into practice in Natal.

During the course of a series of experiments in which the disease was transmitted from sheep to sheep by inoculation, Theiler found that after the tenth passage the virus no longer caused any mortality, and that even the ordinary clinical symptoms were no longer shown, although the temperature chart revealed what might be considered a severe temperature reaction, and the inoculated sheep proved later to have acquired a very satisfactory degree of immunity. He immediately utilized this discovery for practical purposes, and since that date an attenuated virus (defibrinated blood, suitably preserved) has been issued as a vaccine, after careful testing and standardization. The success which has attended the use of the method since its introduction in 1907 has led to its almost universal adoption in the worse-affected districts of South Africa, and the annual demand has surpassed two million doses.

The method is simple and very cheap, and the mortality following its application is negligible, while the immunity produced, although (like that of a naturally recovered animal) not absolute, is sufficient for practical purposes in the very great majority of cases (relapse or reinfection in perhaps 0·1 per cent). Annual vaccination of the whole flock is desirable.

It is advisable to vaccinate as short a time as possible before the Bluetongue season commences, in order to get the maximum degree of immunity at the time of greatest exposure to infection, but of course the vaccination should not be delayed until natural cases actually occur.

In view of the facts that the sheep pass through a fairly severe febrile reaction, and that Bluetongue is a very debilitating disease, certain reasonable precautions should never be neglected, viz.:

(*a*) Sheep should not be vaccinated when in very poor condition, from whatever cause, unless the owner understands the added risk and considers vaccination desirable in spite of it.

(*b*) During the reaction the sheep should not be exposed to inclement weather, and especially to cold rain.

(*c*) Pregnant ewes should not be vaccinated, and

(*d*) Sufficient good grazing should be available to give the sheep every chance of recuperating and recovering condition before the onset of the winter.

It is exceptional for any of the ordinary clinical symptoms to be at all definitely shown by vaccinated animals, and where excessive loss of condition is alleged to have occurred, inquiry will almost always reveal some additional cause, such as the absence of nutritious grazing or, quite commonly, excessive infestation with such parasites as the ordinary wireworm, *Hæmonchus contortus*.

Curative Treatment.—What may be termed good nursing is of considerable importance, and affected animals should be kept isolated in as cool and shady a spot as possible. Soft green food should be supplied to those with sore mouths, and gruel may be administered to any sheep that may be unable to feed at all; plenty of good food should also be given to animals that are unable to stand or walk much, on account of sore feet. The discharges should if possible be cleared away from the mouth and nostrils, especially if respiration is interfered with, and some mild antiseptic or astringent lotion may be applied to the lesions; weak alum solution is commonly used for this purpose. Quinine and various other drugs are often administered internally, but no agent has yet been found to exert any specific action on the course of the disease.

Theiler produced a serum which appeared to possess curative properties, but the use of an efficient method of vaccination rendered the fact of little more than theoretical interest, and he did not consider it to be necessary to continue or to increase its production.

W. H. A.

CONTAGIOUS APHTHA OF LAMBS AND KIDS

Malignant Aphtha; Contagious Ulcerative Stomatitis; Gangrenous Mammitis of Ewes and Goats

This is a contagious disease which usually occurs amongst sheep and lambs or goats and kids when running together, and characterized by an ulcerative stomatitis of the lambs and kids, and gangrenous mammitis of the ewes and goats. There is some doubt as to whether the affection is first introduced by the ewes or by the lambs, but in any case it is usually transmitted from one to the other. The cause of the condition appears to be a micrococcus which may be found in the lesions. The chief method of spread throughout the flock is probably the shepherd's hands, but it is also spread by contaminated grass or other fodder.

Symptoms.—In **lambs and kids** there is a primary fever with refusal to suckle, great debility, and rapid loss of condition. The lambs are dull and listless. On examining the mouth vesicles may be observed on the buccal mucous membrane and the lips. They develop rapidly into ulcers covered with a whitish exudate, and when this is wiped off a red angry-looking base is revealed. The ulcers frequently coalesce and affect large areas, and may involve deeper structures. The ulcers may also extend up the side of the face and become covered with a dry crust. A nasal discharge is sometimes present, while at other times the lungs may become involved or the intestines, setting up diarrhœa. Unless the lambs are fed by hand there is very rapid wasting, and death occurs from exhaustion.

In the **ewes and goats** the mammary gland becomes the seat of a suppurative and gangrenous inflammation. The milk is changed in character, becoming curdy and then blood-stained, and very offensive in odour, the glands enlarged, hot, and painful. Abscesses may form and may burst, emitting a similar offensive material, and frequently a patch of gangrene sets in. In some cases ulcers may be found in the mouths of the ewes as in the case of the lambs. The ewes refuse to feed, and in the early stages are affected with fever, while in the later stages the temperature may become subnormal.

The mortality varies very widely in different outbreaks. Unless the lambs are artificially fed the mortality may be up to 40 per cent, while the mortality in ewes is less if the cases are taken in hand early. If neglected, however, the majority of affected ewes die.

Treatment.—Affected animals should be taken up from the pasture and put into a fold where they are unlikely to contaminate foods. They should be attended by a separate shepherd who should have no communication whatever with the main flock. The food for the ewes should be soft and nutritious, while the lambs must be fed by hand. It is also wise to separate the ewes from the lambs in the affected flock so that infected lambs shall not infect their dams, and *vice versa*.

The stomatitis must be treated by antiseptic mouth-washes such as boracic acid and alum āā ʒi. and water ℥vi. The ulcers might be first painted with tincture of iodine or chloride of zinc grs. v.-℥i. Other useful lotions consist of salicylic acid 1 per cent, or hyposulphite of soda 5 per cent, or permanganate of potash 1 in 1000. In the case of the ewes the udder must be stripped, all discharges collected in a pail and effectually destroyed; they must not be milked on to the ground. The glands should be injected with disinfectant such as warm chinosol solution gr. i.-℥i., or iodine solution 1 per cent. If gangrene sets in the affected part must be removed surgically as soon as possible. Internally they should receive febrifuges and tonics such as magnesium sulphate ℥ss., potassium nitrate ʒss., and powdered nux vomica grs. xv. Stimulants may occasionally be necessary. The use of autogenous vaccines prepared from the udder has been recommended, and useful results have been claimed for this procedure. Subsequently a thorough disinfection of utensils, folds, and buildings must be carried out, and the pastures that have been occupied by affected animals should be left vacant of sheep for the rest of the season.

G. H. W.

NECROSIS OF THE SKIN OF THE HEAD IN SHEEP

This is a condition of sheep somewhat resembling malignant catarrh of the ox, but is known to be due to the necrosis bacillus. It differs from the disease in cattle, in that a number of animals are usually affected at the same time.

Symptoms.—Affected sheep are first noticed to be dull and depressed, and isolate themselves from the rest of the flock. There is a profuse discharge from the eyes, nose, and mouth, while the eyelids become suffused and swollen, and the eyes closed. The sheep refuse food and rumination ceases. The fleece is harsh and dry, respiration blowing, and the temperature is elevated 3° or 4° F. Patches of the skin of the head become swollen and livid, and undergo necrosis. The affected patch may slough off and then exposes a red angry-looking base. Complications may arise, the most common being pneumonia, and the condition then usually terminates fatally.

Treatment.—Separate the affected sheep from the healthy and disinfect the premises in which they may have been. Healthy sheep should be taken out to fresh pastures and examined carefully from day to day, so that affected animals may be speedily removed. Disinfectants and protectives should be applied to the necrotic patches, such as iodine solution 2 per cent, or carbolic lotion 5 per cent, followed by a dusting powder of boracic acid and oxide of zinc. Internally, tonics and febrifuges should be given, such as cinchona or quinine, gentian and iron. Contaminated pastures should be kept vacant for several months.

G. H. W.

CONTAGIOUS ULCERATION OF THE EXTERNAL GENITALS OF SHEEP

Ulcerative Vulvitis in Ewes.—This is a somewhat rare affection occurring in the form of outbreaks in sheep at the breeding season. It is characterized by the development of ulcers of the external genital organs of both ewes and rams, and if not put under control is likely to spread throughout the whole flock.

The *Etiology* of the condition is at present unknown. It is probable, however, that the spread through the flock may occur by coition, and also by the medium of flies. This is supported by the fact that many of the ewes of the flock may be affected although they have not been served by the ram, and the lesions on the vulva are often infested with swarms of flies, which thus easily carry infective material from the lesions and transfer it when they subsequently alight on the vulva of other ewes. This would be more likely to occur if the healthy ewes were in œstrum at the time as the normal discharge would probably be somewhat attractive to the flies. In the writer's experience it has occurred, especially amongst ewes that were docked extremely short, and there was thus no means of warding off attacks on the vulva by flies.

The infective nature of the lesions is shown by the fact that when an ulcer occurs on one side of the vulva the opposing mucous membrane is very likely to become similarly affected, and also that rams become infected after serving affected ewes. In one case lesions on the scrotum of the ram were in direct apposition to similar lesions on the inside of the hocks. The disease dies down in the winter and rarely reappears until the following autumn.

The period of incubation is about three days, a sequence of ulcers being produced during the following eight or ten days.

Symptoms.—In **ewes** the vulva becomes very much swollen and shows angry-looking ulcers, principally round the external margin of the labia. On examining the interior in a few cases other ulcers may be found, but frequently there is only an acute congestion and superficial catarrh of the mucous membrane without ulceration. The ulcers may extend to the skin of the perineum.

In the **ram** similar angry-looking ulcers may be seen at the point of the glans penis and also on the margin of the prepuce. A number of ulcers may also occur on the scrotum, frequently covered with a dried scab under which pus may collect. The skin of the thighs and the inner side of the legs may become affected, apparently by contact with the scrotal lesions. On rare occasions a similar ulcer may be met with on the muzzle of the ram. Under proper treatment the lesions heal in from three to four weeks.

Treatment.—Affected sheep should be immediately separated from the healthy, and the rams removed from the ewes, and service amongst affected stock suspended.

Curative measures consist of disinfection of the affected parts by the application of white lotion or sulpho-carbolate of zinc, gr. v. or vi.-℥i. (one ounce to half a gallon of water). This should be swabbed on to the vulva and other sores, and a little injected in the vagina. After drying, the external lesions may be dressed with on ointment consisting of

Zinci carbonatis ℥ii.
Sol. picis carbonis ℥ii.
Adipis lb. 1
Fiat unguentum.

The worst ulcers may be first lightly cauterized with a silver caustic pencil.

In districts where the disease is known to exist, a careful examination should be made of any new ram purchased during the breeding season. Moreover, it is advisable that too short docking of sheep should be avoided, a proper hygienic condition of the part being maintained by periodical "locking" or clipping of the wool of the parts to prevent soiling, without interfering with the ability of the sheep to remove offending flies.

G. H. W.

CONTAGIOUS AGALAXIA OF THE SHEEP AND GOAT

This is a contagious disease apparently limited to sheep and goats, with lesions principally affecting the mammary glands, eyes, joints, and skin. It is said to be limited to Europe, particularly the mountainous districts of Switzerland, Northern Italy, and Southern France. Sheep and goats of both sexes and all ages are susceptible. It is caused by a filtrable virus, and although contagious it shows no tendency to spread to surrounding districts (an enzoötic). The chief method of infection is by ingestion of food materials or water contaminated with infected milk or discharges from the eyes or from abscesses of the joints or from the skin lesions. It is stated also that the mammary secretion is affected for a considerable time after apparent recovery from the disease. The disease causes great loss of flesh, and in bad outbreaks the mortality may attain 60 per cent. In other outbreaks when the mortality is slight, the great loss of flesh renders the animals worthless for sale or slaughter.

Acute and chronic forms of the disease are recognized.

Symptoms.—The symptoms are both systemic and local. In the acute form there is a sharp rise of temperature, great debility, and loss of appetite, and in some cases death may follow before any local lesions develop. In less acute cases the udder becomes hot, swollen, and painful, and the milk becomes either thick and reduced in quantity, or thin and watery and more copious, and is occasionally blood-tinged. Later the udder undergoes atrophy. The joints of the limbs become swollen and painful, causing great lameness and sometimes resulting in abscess formation. The eye becomes involved with conjunctivitis and opacity of the cornea and a great flow of tears, and the eyeball is very painful. The skin may become affected with pustular eruptions which in the severer cases become confluent.

In the chronic form the systemic symptoms may pass unobserved. The principal lesion involves the udder, which becomes atrophied and soft and on palpation may be found to contain indurated centres. The milk is altered in quantity and quality, and separates into two layers on being allowed to stand. The upper layer is watery with a greenish tinge, and the other layer consists of clots of pus-like material. The joints are sometimes swollen, and may cause temporary lameness. Opacity of the cornea may also occur.

The lambs may be infected *in utero* by infected ewes and show lesions at birth, particularly affecting the joints; the majority of them die. It is stated that lambs born of hyper-immunized ewes are immune to the disease.

Treatment.—Curative measures are not of much service, and treatment is mainly palliative. Isolation and disinfection should be carefully carried out. The udders should be systematically examined, and in the case of a herd of goats kept for milking purposes, the milkers' hands disinfected after milking each goat. No animals should be sold from an infected herd except for slaughter.

Medicinal measures should consist of the administration of a mild saline such as sulphate of magnesia ʒii.-iv., and the local application of mild antiseptics such as saturated solution of boracic acid to the seats of lesions.

As a preventive measure, however, the serum of a hyper-immunized animal is valuable. The duration of the immunity thus conferred has not been proved, but it is sufficiently long to outlast an outbreak of the disease.

Fresh animals introduced into the herd should be isolated for a time and carefully examined with regard to the mammary glands, eyes, joints, and skin, and in the case where the herd is otherwise clean it would probably be economical to slaughter any suspicious animals.

G. H. W.

ENZOÖTIC PARAPLEGIA IN LAMBS

Renguera

Renguera is a serious disease affecting lambs in the Andes regions of Peru, and characterized by derangements of the locomotory apparatus with incoördination of movement leading up to general paralysis commencing with the hind-quarters. It has been studied by Gaiger and others. The disease appears to be contagious in that nearly all lambs born in infected flocks become affected. Lambs up to six or eight months old are most susceptible, and especially so between the ages of two and four months. Yearlings are less frequently affected, and adults very rarely. It chiefly affects animals grazing over marshy pastures, and the sheep improve when transferred to high dry localities.

The course of the disease extends from two weeks to two months, and in the majority of cases terminates fatally, but the older the lamb the greater is the chance of recovery.

The cause of the disease is unknown. A micrococcus has been described as being present in most animals affected, but this organism has not been definitely proved to be the cause of the disease.

Symptoms.—The disease usually begins with a staggering gait, weakness of the hind-limbs, and dragging of the hind-feet, causing abrasion of the skin of the coronets. When standing the legs are wide spread, but when made to move the lamb attempts to do so by jumping, the hind-quarters swaying at the time. If pressed the hind-quarters frequently collapse, causing the lamb to roll over on its side. The course is frequently accompanied by an up and down shaking of the head, and grinding of the teeth. The temperature may be slightly elevated, especially if the animal is disturbed or excited. In the advanced stage of the disease, the lamb is unable to follow its mother to suckle or to graze, and consequently it loses flesh very rapidly and ultimately dies of inanition.

Anatomical Changes.—The lesions to be found in lambs dead of renguera are not well marked, and, beyond the emaciation of the carcase, might easily be overlooked. The principal changes include an increased amount of liquid in the serous cavities slightly blood-tinged, the liver darker in colour than normal, sometimes catarrhal enteritis, slight congestion of the meninges of the brain and spinal cord, and in a number of cases patchy endocarditis.

Treatment.—No curative measures have been found serviceable. Preventive measures consist in the isolation of infected flocks, and the immediate slaughter of all affected animals. A vaccine has been prepared from the micrococcus already mentioned, but its efficacy is at present doubtful.

G. H. W.

TAKOSIS IN GOATS

Takosis (wasting disease) is a chronic contagious disease occurring amongst goats, and characterized by progressive anæmia and emaciation. It has been described as specially occurring amongst Angora goats in America and in South Africa, but an affection apparently closely corresponding to it has frequently been observed also in Europe affecting other breeds of goats. It is possibly the same disease as that described by Pegler in the British Isles under the name of "a disease peculiar to goats."

Etiology.—Takosis is believed to be due to a Gram-positive organism called by Mohler *Micrococcus caprinus*. The organism has been found in the blood and also in the spleen, pericardial fluid, and the kidneys.

Symptoms.—The first symptoms noticed are a gradual loss of condition associated with listlessness. The goat becomes languid, its ears become drooping, and it tends to separate itself from the rest of the herd or to lag behind when being driven. The condition is very slowly progressive, and the goat usually eats and ruminates fairly well until the condition is far advanced. The temperature may be elevated to 105°-106° F. in the early stages, but later becomes subnormal. The back is arched, flanks hollow, and the abdomen somewhat pendulous. As the condition advances an offensive diarrhœa often supervenes, and an occasional cough and slight nasal discharge with snuffling may be noticed. The eyes become sunken and the mucous membranes very pallid, and the goat ultimately can only rise with difficulty, staggers, and frequently falls, the condition passing on to more or less complete paralysis.

The course extends from one to three months, the animal ultimately dying from inanition. Recovery of an animal having shown symptoms is practically unknown.

Pathological Anatomy.—On post-mortem

examination the most noticeable feature is extreme emaciation and anæmia. The bowels are in a state of chronic catarrh and may show small areas of superficial necrosis ; the contents may be either normal or semi-liquid. The lungs are mottled—pink, dark red, and grey—but there is little or no consolidation. The liver and heart muscle are paler than normal, and there is a pinkish pericardial fluid.

Differential Diagnosis.—From the clinical symptoms this disease appears to be quite indistinguishable from Johne's disease, and also from parasitic gastro-enteritis. The writer is of opinion that most of the cases described as takosis in the British Isles are in all probability cases of Johne's disease. Positive diagnosis may be arrived at by examination of the fæces for eggs of parasites, or for bacilli, or by testing with avian tuberculin, or, when that fails, by the slaughter of one of the worst affected and post-mortem examination.

Treatment.—As already indicated, most cases of takosis terminate fatally, from which it will be realized that there is no satisfactory curative treatment known. The course of the disease, however, may be checked when taken in hand early by attention to diet, which must be dry and highly nutritious.

Medicinally, Mohler and Washburn claim the best results from calomel, gr. iss. given twice daily for two days followed on the third day by white arsenic, gr. i., sulphate of iron or reduced iron, gr. ix., and sulphate of quinine, gr. iv. This is repeated night and morning for ten days, and after two days' interval the course is repeated. Affected animals should be taken from the pasture in order to avoid further spread of the disease.

Preventive measures consist solely in avoiding the introduction of affected goats into the herd and the maintenance of good bodily condition by generous diet in order to fortify the animal against any infection. Isolation or segregation should be immediately carried out when any wasting disease has been observed in the herd.

G. H. W.

INFECTIOUS PNEUMONIA OF GOATS

This disease occurs in various parts of the world, and has especially been seen in Angora goats. It is caused by a bacillus with bipolar staining, and is undoubtedly a form of hæmorrhagic septicæmia as affecting the goat. (See "Hæmorrhagic Septicæmia.")

INFECTIOUS PLEURO-PNEUMONIA OF GOATS

An acute form of pneumonia and pleurisy has been described as occurring in various parts of Europe, particularly in the mountainous districts of Germany, Italy, and the Pyrenees. and also in Algeria. It has also been met with in India. It appears to be limited to goats, and is not contagious to sheep or cattle. The disease runs a mild course in animals born in indigenous areas, but it assumes an acute and severe course in animals newly introduced into the district.

The period of incubation is from eight to ten days.

Etiology.—The causal organism of this condition has not yet been discovered, but circumstances point to the probability that it will ultimately prove to be a filtrable virus.

Symptoms.—The symptoms commence with blowing respiration and a cough, and the temperature is elevated several degrees. In acute cases dyspnœa sets in early owing to a large area of the lung being involved, and the appearance of a considerable quantity of inflammatory liquid in the pleura. In very acute cases the attack proves fatal within twenty-four hours. In the less acute cases an eruption may develop on the lips and the udder, and the animal becomes very weak and emaciated. The course in such cases extends over three or four weeks.

Pathological Anatomy.—The condition of the lungs and the pleura somewhat resemble contagious bovine pleuro-pneumonia. There is a sero-fibrinous exudate into the chest, with a deposit of fibrin on the pleura. When the affected area of the lung is cut across the interlobular septa are seen to be distended with a similar exudate.

Treatment.—No curative measures have been found of any service, and the best course to pursue is to slaughter all affected animals and in contacts. A strict quarantine should be established, and thorough disinfection resorted to.

G. H. W.

MALTA FEVER IN GOATS

This disease is now generally termed "Undulant Fever," but it has been known for a long time under the names of "Malta Fever," "Mediterranean Fever," and "Rock Fever."

As far as the domesticated animals are concerned, practically all the different species are susceptible, and in one species (the goat), a large number of individuals in certain areas are infected. Thus it was found in Malta that 50 per cent of the goats on the island were infected with the disease.

The infected animals are so little affected in their general health, however, that the condition would hardly have received any attention, or have been recognized as a specific disease, but for the serious character of the disease in man, and the undoubted importance of infected animals in carrying and transmitting the causal organism.

The disease may be described as a septicæmia, as the causal organism is found in the blood stream, and may often be cultivated from blood (of man) withdrawn during life.

Distribution.—Undulant fever in man has long been known to occur in Malta and Gibraltar and in various other centres along the Mediterranean coast, but in recent years, with the improved methods of diagnosis, it has been shown to have a very wide distribution in tropical and subtropical countries.

Apart from its wide distribution in the majority of the countries bordering on the Mediterranean Sea, the disease has been recorded from India, China, North, East, Central, and South Africa, several South American states, and the United States of America.

Etiology.—The causal organism, *Micrococcus melitensis,* was isolated by Bruce in 1887. It is a small, non-motile, rounded or slightly oval coccus or cocco-bacillus, measuring about 0·4 microns in diameter, and generally occurring singly or in pairs.

It may be stained with all the ordinary basic aniline dyes, such as methylene blue, but does not retain the stain in Gram's method.

The organism is ærobic (facultative anærobe) and grows best in slightly acid or only faintly alkaline media; probably the most favourable medium for its growth is 5 per cent glycerine agar. The optimum temperature is 37° C., and growth is very slow, but some growth occurs at ordinary room temperature. Primary cultures are usually obtained from spleen pulp spread on sloped agar.

Post mortem the organism is found to be particularly abundant in the spleen, but it is also found in the liver, kidney, etc.

The organism is fairly resistant outside the body, and has been shown to retain its vitality for a month in water and for more than two months in dust or dried sterile manure. On the other hand, it is easily killed by heat and disinfectants.

It appears that some cases which would be classed as cases of Undulant Fever are caused, not by *M. melitensis,* but by certain very closely allied organisms which have been distinguished from that species by biological tests.

Susceptibility.—Besides the goat, which is very commonly infected in the endemic centres, horses, mules, donkeys, cattle, sheep, dogs, and fowls have all been shown to be liable to natural infection, but, as has been stated, the disease owes its importance to the susceptibility of man.

Method of Infection.—It has been shown that the milk and urine of infected animals contain the micrococci, often in large numbers, and the fæces may occasionally be infective. The investigations in Malta of the Commission on Mediterranean Fever in 1904-5 led to the conclusion that human cases very commonly arise as a result of the ingestion of the milk of infected goats. This conclusion was subsequently supported by the large measure of success obtained in the prevention of the disease by avoiding the use of goat's milk or of milk which had not been sterilized. Milk products, such as cream and cheese, may also harbour the organism and prove infective.

Apart from the ingestion of infected goat's milk and its products, it is probable that cases in man also arise from contamination of the food by dust, from soil on which the urine of infected animals or men (ambulant cases) has been voided.

Amongst animals the infection is probably acquired, in the majority of cases, by the ingestion of food materials contaminated by the urine and fæces of infected animals, or by dust from surface soil so contaminated. Infection may be carried from goat to goat during milking.

Infection by inhalation has been proved to be possible under experimental conditions, and infection through the skin (inoculation into skin lesions) and mucous membranes may occur, but the common channel of entrance is undoubtedly the alimentary tract.

It has been suggested that transmission during coitus is common, and that the he-goat plays an important rôle; in an infected flock the entire males may be infected in a greater proportion than the females.

Symptoms.—In the majority of cases goats (and other domestic animals) show no perceptible signs of ill-health as a result of infection, and they may remain infective for a long time, even for years, without exhibiting any symptoms of general disturbance.

Goats found to be excreting very large numbers of *M. melitensis* in the milk often appear to be in poor condition, and it is said that a chronic mastitis is occasionally observed, but these conditions are not in any way constant, or of diagnostic value.

Certain changes in the appearance and other properties of the milk have been described in various cases, but they have also proved to be not at all constant; a sub-acute or chronic bronchitis, causing coughing, is seen fairly frequently.

The investigations of several authors have shown the importance of the frequency of abortions as indicative of the presence of the disease in a flock of goats, and, according to some authors, 50-90 per cent of the pregnant animals may abort when the disease first appears in a herd. Abortion generally occurs in the fourth month, but may rarely occur as early as the second, and the fœtus may be carried to full term, but is then usually born dead, or lives only for a short time. The act of abortion is generally preceded only by indefinite symptoms, especially at the early stages of pregnancy, and the fœtus appears normal.

Abortion is not rarely followed by such complications as catarrhal vaginitis, which generally disappears in about forty days, an obscure lameness, and a mammitis associated with the formation of nodules of about the size of hazel nuts, which is generally not serious, and recovers without treatment.

The goat may fail to carry to full term at the second, and even the third pregnancy after infection.

In he-goats lameness may occur, and very rarely orchitis. Sheep are said to be fairly commonly affected in the infected districts of France, and the symptoms closely resemble those seen in goats, abortion being the most important.

In man there are some "ambulant cases," showing few or no symptoms of ill-health, and a mild intermittent type of fever may also occur, but in typical cases there is an attack of fever, with a temperature rising and descending somewhat gradually, followed by a long series of relapses and intermissions extending over two months, or possibly much longer. During the relapses there are painful but transient joint affections, neuralgic pains, and occasionally orchitis. Anæmia and mental depression make their appearance later.

There are also malignant cases with high continued fever, aggravation of the usual symptoms, and often pulmonary complications.

Post-mortem Lesions.—In the domestic animals fatal cases are almost unknown, and post-mortem findings are therefore not of importance.

In man, and in experimental animals which have succumbed to the disease, the most prominent lesion is enlargement and acute congestion of the spleen. There is some congestion of the liver, kidneys, lungs, and alimentary canal; in some cases there is even ulceration in the colon.

Diagnosis.—The clinical diagnosis of Malta Fever, which is often very difficult in man, is generally quite impossible in animals, and recourse must be had to laboratory methods.

A bacteriological diagnosis, based on the isolation of the specific micro-organism from the milk or urine, may be made, but in practice diagnosis is almost invariably based on the result of tests carried out with the blood serum.

The agglutination test, which was shown by Wright to be a suitable method for the detection of the disease in man, has also proved very reliable for the diagnosis of infection in goats, although it is said that occasional failures to indicate the presence of infection occur with this method.

Mohler and Hart, working in the United States, found that the serum of healthy goats may agglutinate *M. melitensis* in a dilution of 1 in 40, and an agglutination within $1\frac{1}{2}$ hour with a dilution of 1 in 70 was therefore fixed by them as the standard for a positive reaction; agglutination with much lower dilutions, such as 1 in 30, has been claimed by some authors to justify a positive diagnosis. The agglutination test can also be applied to the milk.

It has been shown by Mohler and Eichhorn that the complement-fixation test can be utilized for the diagnosis of Undulant Fever in goats, and these authors suggest the use of the complement-fixation test in conjunction with the agglutination test.

Prophylaxis and Treatment.—In man sensitized vaccines are said to have given good results, and possibly such a method may become applicable to goats in countries in which the infection is widespread.

In localities where only a small proportion of the goats are affected the best measure to adopt is undoubtedly the slaughter of all infected animals, which could be detected by periodically repeated serum tests.

Thorough disinfection of infected pens and utensils and attention to the water-supply and general sanitation are indicated.

Where the infection is prevalent, personal cleanliness is important for the protection of human beings; local milk products should be avoided, and the milk should be boiled or pasteurized (*i.e.* heated for twenty minutes at 145° F.).

Goat pens, etc., should not be close to human dwellings or the water-supply.

W. H. A.

RABIES

Canine Madness; Hydrophobia (in Man)

Rabies is a specific contagious disease, principally affecting dogs but inoculable to almost all species of animals, and characterized by acute derangement of the nervous system, causing interference with consciousness and producing nervous irritability and paralysis. Although the disease may affect almost all species of animals it should properly be regarded as a canine disease since it is mainly through animals of the dog tribe, both domesticated and wild, that the disease is maintained and spread.

Rabies has a very wide distribution throughout Europe, Asia, Africa, and America, but it has not yet been introduced into Australia or New Zealand. There is nothing peculiar in those countries that would in any way prevent the rapid spread of the disease if an affected dog was introduced and allowed to run free. The disease was stamped out of the British Isles where no cases occurred between 1902 and 1918. This was due to the courageous enforcement of very energetic measures which included the muzzling of all dogs in public places, the institution of a six months' quarantine of all imported dogs, and the destruction of all stray dogs. In 1918 to 1921 a rather serious outbreak occurred in the south and west of England as the result of an infected dog having been smuggled in during the incubative stage of the disease.

Etiology.—Rabies is caused by a filtrable virus which is inoculated into the tissues through a wound usually inflicted by the teeth of an affected animal, commonly a dog. The virus is present in the saliva of rabid animals, and infection may occur through any wound soiled with such saliva. The virus travels up the nerve trunks to the brain and spinal cord where it multiplies and accumulates in great concentration. It may also be found in the nerve tissues of other parts of the body, having been disseminated through the nerve trunks and not by the blood stream. The virus has been cultivated by Noguchi, and in the cultures small rounded or oval granular bodies were observable, just visible with a magnification of from 1000-2000. They vary in size from one to twelve microns. Inoculation of these cultures produced rabies both in dogs and the smaller experimental animals. It can thus no longer be regarded as an ultra-visible virus. The virus can only multiply naturally within the animal body, but it may remain infective a considerable time after the death of the animal, and can withstand putrefaction. Dry air attenuates it.

The virus of rabies may be increased in intensity by passing through a series of rabbits, while it may be diminished by passing through a series of monkeys. When virus taken from a naturally occurring case in a dog is inoculated into rabbits the period of incubation is about fourteen days, and it usually proves fatal on the seventeenth day. This degree of virulence is called " street " virus. When, however, it has been passed through a series of about 50 rabbits it will kill them on the seventh day. This period may be reduced to five days by using young rabbits. The intensity of the virus cannot be increased beyond this point, which is therefore called " fixed " virus.

The natural method of infection is through a wound, but it may also be transmitted through an uninjured mucous membrane, as of the nose and eye. The saliva of an affected animal has been shown to be virulent several days (as long as fourteen days) before the appearance of symptoms. The danger of infection is greatest when the bite is inflicted directly through the skin, and especially if near the head. The danger is rather less when the bite is through clothing or through a thick coat of hair or wool. These frequently remove the saliva and virus from the teeth before the skin is actually wounded. In some cases the virus is carried away by bleeding of the wound or destroyed by immediate disinfection of the wound. It is believed that probably somewhat less than one half of the animals bitten by rabid dogs contract the disease, and that of the total number of human beings bitten by rabid dogs about 16 per cent develop rabies, and die. This low percentage is due to the passing through the clothes. Of human beings bitten in the face by rabid dogs the mortality is about 80 per cent.

The period of incubation is very wide and varied, and ranges from nine days to fifteen or eighteen months. The latter periods, however, are very rare. The average period in dogs, sheep and swine, is from fifteen to sixty days, while in horses and cattle it is usually a little longer, averaging from thirty to ninety days. It is shorter if infection has taken place near to the head, and it is also reduced in young animals.

Rabies in the Dog

It is common to describe two forms of rabies, viz. *mental* or *furious rabies*, and *dumb* or *paralytic rabies*. There is probably no fundamental difference between these two forms, the difference being that in the former case the period of excitement is very pronounced and rather prolonged, while in dumb rabies the period of excitement is very short, and paralysis, especially of the jaw and throat and of the hind limbs, sets in unusually early.

The *symptoms* may best be described in three stages.

(1) **The Stage of Melancholy.**—This stage is often unobserved, and consists merely of an alteration in habits of the dog. There is general dullness and depression, and some little indifference to his master, sometimes associated with general uneasiness, scratching with the feet and a tendency to hide under the table or in dark corners. A usually noisy dog may become quiet and docile, whereas the placid dog often becomes excitable. This condition may last from one to three days and gradually merges into the second stage.

(2) **The Stage of Excitation or Irritation.**—By this time the dog becomes rather more excited, unable to recognize his master, and appears

often to have some difficulty in swallowing. At the same time he snaps at imaginary objects and jumps in an excited manner at any unusual noises and on being touched. The eyes appear to have a vacant expression and the pupils are dilated. There is a peculiar change of appetite, which becomes morbid—the dog frequently refusing all normal food, but eating foreign bodies such as straw, carpets, wood, stones, horse dung, etc. If in confinement the dog is constantly attempting to get out, and as soon as he is free he usually runs away from home, running ahead quite aimlessly, going for miles even on the hard road. Rabid dogs rarely return to their own home. Any other animals or human beings met on the way will usually be attacked and bitten, but rabid dogs will rarely go out of their way to make attacks, and they are not likely to attack human beings unless molested by them. Some dogs even bite themselves, particularly if kept in confinement. During this stage there is often increased sexual excitement. There is no dread of water and no disinclination to drink, consequently the term "hydrophobia" should not be given to rabies in dogs. This stage of excitement, which may last from three to four days, gradually passes into exhaustion and develops into the third stage.

(3) **The Stage of Paralysis.**—In this stage weakness of the hind limbs develops, and the dog may stagger and fall. This passes on into general paralysis. The muscles of the jaw become affected, and the lower jaw drops, causing dribbling, and often frothing at the mouth and increased difficulty in swallowing. The larynx may be thrown into spasms, producing an alteration in the voice. In attempting to bark the animal emits a peculiar howl which is in many cases said to be diagnostic. As the condition advances the patient becomes extremely weak and emaciated, and there is progressive incontinence of urine and fæces, and death ultimately occurs in from four to eight days after the onset of symptoms. Recovery from rabies is exceedingly rare, but recoveries have been observed by Pasteur and others.

Diagnosis.—Diagnosis may be arrived at by a combination of methods, viz. from the clinical symptoms observed, from the conditions discovered on post-mortem examination, by microscopic examination of parts of the nervous system, and by experimental inoculation of rabbits or guinea-pigs. Neither of the first two conditions affords a basis for making a positive diagnosis, although one may justifiably express a strong opinion on the probability of the case.

Clinical Diagnosis.—In order that a careful observation may be made of the symptoms, a suspected dog that has bitten a human being or other animals should never be slaughtered. It should be placed under control so that it can be carefully observed, and the disease allowed to run its natural course. This procedure undoubtedly facilitates diagnosis of the condition, for if the affection be rabies it will prove fatal in three or four days. If the dog recovers from the illness giving rise to the suspicions, or survives for six or eight days, the condition may be pronounced "not rabies."

Post-mortem Examination.—The feet of most dogs dead of rabies usually show signs of the animal having travelled a considerable distance. The mouth and pharynx are usually congested and often present small wounds due to the dog having attacked foreign bodies. The condition of the stomach is of great importance. In the vast majority of cases it is in a state of congestion, and may show small hæmorrhages. It is almost invariably devoid of normal food material and contains instead foreign bodies such as straw, bits of carpet, wood, stones, etc., if the animal has been at liberty.

Microscopic Examination.—This resolves itself into an examination (1) for the detection of changes in the nerve cells of the sympathetic ganglia, which are quite peculiar to and characteristic of rabies, and (2) for the detection of Negri bodies in certain parts of the brain.

The change occurring in the sympathetic ganglia consists of a degeneration of the nerve cells and proliferation of the cells of the capsule which thus compresses the nerve cell. For this examination the plexiform ganglion of the vagus nerve is the one usually selected. Discovery of these changes on microscopic examination may be regarded as positively diagnostic of rabies, but inability to demonstrate them does not justify an absolutely negative diagnosis.

The examination for Negri bodies consists of making either sections or impressions of preparations from certain parts of the brain, preferably the hippocampus. By a special method of staining, peculiar corpuscles may be found occupying the nerve cells of the part. They vary in shape, sometimes round, oval, pear-shaped or angular bodies, varying in size from half a micron to twenty-five microns, the small ones showing no differentiation of structure while the larger ones appear to contain small enclosures of varying shapes. These Negri bodies are found in 96-97 per cent of all cases of rabies, and when present may be regarded as absolutely diagnostic of rabies since they have not been proved to exist in other diseases. Inability to discover them, however, does not justify the opposite conclusion. The discovery of Negri bodies enables a positive diagnosis to be made within a few hours after death, and moreover, they are very resistant to putrefaction, and can consequently be demonstrated in nerve tissue after decomposition has set in. It has been suggested that these bodies are organisms belonging to the class Protozoa, but that view is by no means generally accepted.

Experimental Inoculation.—This is the most certain method of diagnosis. In all cases where the brain of the animal has been obtained before becoming putrid, the material for inoculation is taken from the medulla oblongata of the suspect and is introduced into the cranium between the dura mater and the surface of the brain of rabbits with sterile precautions. If the animal is rabid the rabbits develop rabies in about fourteen days, commencing with a paralysis of the hind limbs. The paralysis soon becomes general and the affected rabbit usually dies in about seventeen days after inoculation. Affected rabbits do not become aggressive and show no tendency to bite, and can therefore be handled with a minimum of danger. If rabbits die at an earlier stage, it is generally due to the putrid condition of the inoculating material. In those cases where the brain substance is suspected of decomposition, it should be placed in glycerine for a week or more. This destroys the putrefactive organisms, but apparently does not have any deleterious effect on the virus of rabies within two or three weeks. The great objection to this method of diagnosis lies in the time required (about seventeen days) before diagnosis can be made. In the case of persons bitten by suspected animals the other methods of diagnosis should first be resorted to in order that the victim may undergo special preventive treatment for rabies in the event of a positive diagnosis, otherwise the person bitten on the hands or face may actually develop rabies and die in less time than would be the case with the inoculated rabbits.

Method of Dispatching Specimens for Diagnosis.—In cases where the observation of the symptoms and post-mortem examination have failed to reveal a definite diagnosis of "not rabies," a portion of the dog should be sent with all despatch to a laboratory for diagnosis. If it is not possible to send the whole body, the head should be removed with the skin intact by cutting through rather below the middle of the neck. In this way the plexiform ganglia are left intact for microscopic examination if it reaches the laboratory within twenty-four hours. It should be wrapped in a clean cloth, and if possible in warm weather packed with ice in a box for dispatch. If the specimen is likely to be more than twenty-four hours before reaching the laboratory it should be immersed in glycerine, or the brain, including the medulla, carefully removed and packed in glycerine and sent to the laboratory. In the case of large animals the brain with the medulla should be packed and sent in a similar manner with all dispatch.

Differential Diagnosis in the Dog.—The following conditions have from time to time been confused with rabies in the dog:

Epilepsy.—This condition has been suspected by laymen owing to the development of peculiar habits on the part of the patient, and convulsions affecting various parts of the body, frequently causing frothing at the mouth. It is easily distinguished from rabies by the sudden complete loss of consciousness, and the varying intervals between the attacks. Moreover, the condition is rarely fatal, while in rabies death usually occurs within five days after the onset of symptoms.

Ascarides or Round Worms in young dogs frequently set up symptoms of frenzy and convulsions, often more resembling epilepsy.

Injury to the Jaw.—Fracture of the lower jaw, or softening of the bone or dislocation of the jaw, each cause dropping of the lower jaw and dribbling. In the two former cases the jaw can be easily closed, and may be confused with "dumb" rabies. The history of the case, and the fact that the suspect survives the period in which rabies usually proves fatal, enables one to differentiate. In the case of dislocation the jaw is usually wedged open but may be reduced. In other respects the differentiation is made as in the case of paralysis.

A **bone fixed between the upper incisors and molar teeth** across the inside of the mouth frequently causes a dog to froth at the mouth, and to rush about in an excitable condition. He dribbles at the mouth and snaps at any one attempting to restrain it, and thus the condition may be confused with rabies. It can generally be distinguished, however, by the frequent shaking of the head and by the animal rubbing his face along the ground and scratching at his face with his paws in attempts to dislodge the foreign body. The discovery of the foreign body of course puts an end to any confusion.

Foreign bodies further along the alimentary tract such as in the pylorus and duodenum, or even in the rectum, will often cause dogs to become frantic. Such cases, however, are rarely fatal within the same period as rabies, but if so a post-mortem examination gradually reveals the cause of the trouble.

Rabies in Cats

Cats are not often affected with this disease. When they are affected, however, the symptoms closely resemble those of the dog. They are very aggressive, attacking both animals and man, especially attempting to injure their faces with teeth and claws. They emit a peculiar howl, and develop muscular spasms terminating in paralysis of the hind limbs. The course of the disease in cats is often shorter than in the dog and extends from two to six days.

Rabies in Cattle

In cattle the disease is almost always produced as the result of being bitten by an affected dog, and is rarely or never spread from cow to cow.

Symptoms.—General uneasiness and excitement, stamping and scraping the ground, and a general disposition to do damage. There is frequent bellowing, muscular twitchings and dribbling of saliva. The appetite is depraved, and rumination and lactation are suspended. There is frequently constipation and straining or tenesmus which may give rise to diarrhœa: Tympany and sometimes vomiting may occur. There is increased sexual excitement, great loss of condition, and exhaustion followed by paralysis commencing with the hind limbs. In some cases the excitement stage is very short or absent, and the symptoms then more closely correspond to the so-called dumb form of rabies.

Rabies in cattle usually terminates fatally in from three to six days.

Similar symptoms have been observed in deer.

Rabies in Sheep and Goats

In sheep and goats rabies sets up symptoms very similar to those in cattle. In each case the animals have a tendency to become excited and aggressive, attacking practically any class of animal, even dogs. In many cases, however, the excitement stage is either very short or wanting, the stage of depression and paralysis being much more pronounced.

The course is about the same as that for cattle, and usually proves fatal in from three to five days.

Rabies in Swine

In pigs the symptoms of rabies very closely resemble those in the dog. General irritability is first noticed, together with nervous excitement. They become aggressive, and this stage quickly passes on to paralysis and proves fatal in from one to four days.

Rabies in Horses

In horses the stage of irritation is very marked, the animal breaks out into patchy sweats and walks round his box or stable with a tendency to bite or rub the seat of inoculation. Affected horses frequently paw the ground and have a staring eye, and often dribble from the mouth. When there has been no history of a bite from a rabid dog such cases are sometimes first diagnosed as colic. There is increased sexual excitement and frequent micturition. Affected horses generally refuse food, but will bite at the manger or other objects available. They are usually very thirsty, but have some difficulty in swallowing. When attempting to neigh the change of voice may be noted. The temperature may be a little elevated but not very pronouncedly so. The symptoms of excitement are followed by depression with paralysis affecting the muscles of the throat and of the hind limbs, and death usually occurs in from three to four days after the onset of symptoms.

Rabies in Man

In the human being the course of rabies may be described in three stages as in the case of the dog. In the first stage there is the period of *general malaise* with depression and indisposition, pain at the seat of inoculation, with swelling of the lymphatics leading from the part. There is frequently some stiffness, especially in the region of the joints, and an elevation of temperature, and in some cases where there has been no history of a bite the diagnosis of acute rheumatism has been made. During this stage there is a disinclination to drink. This passes on into the second or *hydrophobic stage*, during which the patient has great dread of water; both the sight, sound and touch of water are apt to produce convulsions, particularly in the region of the throat. Spasms of the larynx frequently occur, causing the patient to emit peculiar sounds, which have been stated to resemble the bark of a dog, but which in reality have no resemblance at all. These paroxysms are very easily set up with slight disturbance, and may be associated with hallucinations, often with a tendency to become aggressive. This passes gradually into the third stage of exhaustion and *general paralysis*, which soon terminates in death.

Treatment.—When rabies has once developed there is no known treatment that will influence the course of the disease or effect a cure. In such cases the affected animal should be put under effective restraint in order that the course of the disease can be properly observed for a positive diagnosis to be made. When a positive diagnosis has been arrived at all animals that have been bitten by an affected animal should either be slaughtered or be placed in rigid quarantine for not less than three months. Since the period of inoculation often exceeds that period, it would perhaps be advisable where practicable to extend the period of quarantine to six months, especially in the case of animals of the dog and cat tribes which use their teeth as weapons of offence. Food animals that have been bitten by a rabid dog and slaughtered before any symptoms have developed may with safety be used for human food. Human beings bitten by a rabid dog should at once undergo immunization by one of the several methods whose efficacy has been well established.

In the British Isles rabies is scheduled under the "Contagious Diseases (Animals) Act," and any outbreak or suspected case must be at once reported to the police. Steps are then taken to verify or negative the suspicion, and all animals that have been bitten by the suspect or have been exposed to infection are quarantined. If the diagnosis is confirmed all animals that have been bitten are slaughtered, and no dogs are allowed in a public place unless effectively muzzled and under control, while stray dogs are seized and destroyed. In order to prevent the introduction into a non-infected country strict quarantine should be enforced for from three to six months of all carnivorous animals imported from infected countries.

Preventive Inoculation.—This consists of the injection into animals or man of very minute doses of virus obtained from the spinal cord of rabbits which have been killed by "fixed" virus. It has been shown that such cords may be attenuated by desiccation by suspension in a stoppered jar over caustic potash. This is the method adopted in the Pasteurian system, which is the one most frequently adopted.

Pasteurian Treatment of Persons bitten by Rabid Animals.—Pasteur was able to show that spinal cords of rabbits killed by "fixed" virus lose their virulence after desiccation over caustic potash for fourteen days at a temperature of 23° C., and that cords exposed in a similar manner for a shorter period were also attenuated in an inverse proportion to the time during which they had been desiccated. He found also that he could protect dogs and other animals against rabies by first injecting emulsions of the weak cords and gradually working up to the strong cords, and by this means he was able to anticipate the action of the virus introduced by a bite, and thus confer immunity before that virus had reached the brain and set up the disease. His method, therefore, consists of subcutaneously injecting a person bitten by a rabid dog with an emulsion prepared from pieces of two cords desiccated for fourteen and thirteen days respectively, and day by day a stronger cord is taken until the treatment is completed with a cord that has only been desiccated for three days. The inoculation with cords dried five and three days is then repeated once or twice. The whole treatment lasts about fifteen days.

In the case of people bitten about the head and face the treatment is prolonged for three weeks, and the stronger cord is more rapidly worked up to so that a three days' cord is used as early as the seventh day For the next fortnight cords dried for five, four and three days respectively are alternated.

The success of this method is somewhat remarkable, and statistics go to prove that of all persons bitten by rabid dogs and treated at the Pasteur Institute less than 1 per cent have died from rabies, while of persons bitten about the head and face by a rabid dog the mortality has been reduced from 80 per cent without treatment to less than 4 per cent with treatment. The success of the method must also depend upon some extent with the rapidity with which it is carried out after being bitten, so that persons undergoing the treatment should submit themselves at the earliest possible moment for inoculation. The immunity produced by preventive inoculation lasts for one or two years.

Hogye's Method.—This consists of the inoculation of vaccine prepared from the spinal cords of rabbits killed with "fixed" virus in very minute doses, and injecting them subcutaneously every few hours in gradually increasing doses. This method seems to show that the important feature of the treatment consists in the quantity of the virus used rather than its attenuation, and it has been suggested that the effect of desiccation of the cords for the Pasteurian method is to reduce the quantity of virus in the cords rather than to reduce its intensity. It is also suggested that although the passage of rabies virus through a series of rabbits intensifies it for that animal it probably attenuates it for the human being.

Another method (Galtier's) of preventive inoculation consists of the intravenous injection of virus obtained from the brain substance of rabbits dead of "street" rabies. This method is still in the experimental stage, but good results are claimed for it.

G. H. W

CANINE DISTEMPER

Catarrhal Fever of the Dog; Canine Plague; Canine Influenza

Canine distemper is a specific contagious disease chiefly affecting young animals of the canine tribe and characterized by fever, catarrh of the mucous membranes and frequently nervous derangements. Distemper has a world-wide distribution and often occurs in the form of epizoötics over wide areas. As already stated it especially affects young dogs, most of which contract the disease within their first year of life. Adults, however, are susceptible, the relative infrequency with which they are affected is due to immunity partial or complete which has been acquired as the result of recovery from an attack in early life. Immunity thus developed is, however, not absolute, and it is by no means uncommon for animals to be affected a second time in later life, but in such cases the attack is generally, but not invariably, much milder than in the case of the first attack. There is a popular idea that all dogs must of necessity have distemper. That is only true in so much as the disease is so widespread that it is almost impossible for an animal to pass through life without exposure to infection, and so far as we are aware natural immunity does not exist in any type or age of dog. Animals that escape the disease only do so by virtue of the fact that they have not been exposed to infection, while frequently other animals stated not to have had the affection have in reality been affected with distemper in a very mild form which may have escaped observation. In one of the latter cases one can frequently ascertain on making enquiries that at some date the dog did have a " slight cold and cough," but as it was so mild the owner did not regard it as being distemper.

Etiology.—The causal organism of distemper has not been positively made out, though a number of organisms have from time to time been described as the cause of the disease. It is most probably due to a filtrable virus which sets up a primary fever, catarrh and debility, and thereby permits the invasion of various parts of the body by a variety of organisms more or less constantly present in dogs. These organisms are therefore secondary, and are largely responsible for the varied clinical pictures presented during the course of an outbreak of distemper. The most important secondary invaders are the *bacillus bronchisepticus canis* in the respiratory tract, small *bacilli of the colon type* in the alimentary tract, and *streptococci* and *staphylococci* in the cutaneous lesions. The virus is contained in the various discharges in the early stages of the attack. Natural infection takes place either by direct contact of the susceptible animal with an affected one or indirectly through ingestion of food or water contaminated with discharges. It must also be admitted as the result of numerous clinical experiences that certain premises may become practically hot-beds of infection. This applies especially to various dogs' homes or kennels and to dealers' establishments. Such places are rarely free from the disease, and in London at any rate it is usually expected that any susceptible dog having been for a few hours in such places will contract the disease in a more or less severe form. It is rather difficult in such cases to state positively whether it is the premises that are actually infected, or whether infection is maintained only by the frequent interchange and traffic in dogs. The latter is the more probable explanation and probably also accounts for the frequency with which dogs develop distemper after various shows. It is improbable in such cases that the show benches which may be used only once or twice a year would remain infective after such intervals.

Amongst *predisposing causes* to distemper must be included youth and exposure to inclement conditions such as spring and autumn weather. Various breeds have been said to be more susceptible to distemper than others, but no breed is exempt. It appears to be a fact, however, that finely bred dogs of any breed are, largely owing to inbreeding, more susceptible than coarsely bred dogs or mongrels. It is often thought that the various short-nosed varieties are more susceptible than others. That is somewhat doubtful, though it must be admitted that when once affected many of those animals appear to have less resistance against the disease.

The *period of incubation* of distemper in natural cases varies from three days to three weeks, the average being ten days. In cases experimentally produced by hypodermic injection of filtered virulent nasal discharge or pericardial fluid, the period of incubation is reduced to three or four days.

The *course* of the disease is very varied, depending largely upon the complications which may have arisen. The average course in uncomplicated cases is about three weeks (two to four weeks), but various complications may extend the period to several months.

Anatomical Changes.—The morbid changes met with in distemper vary considerably with the case. There will usually be found a purulent discharge from the eyes and nose and the presence of a number of flat pustules from ¼ in. to ½ in. in diameter in various parts of the skin, particularly where the hair is thin or absent, such as under the abdomen, inside the thighs and the axilla. The eyes may show opacity of the cornea, often with ulceration and even ophthalmia. In very acute cases there is only a slight watery nasal discharge with some congestion of the mucous membrane of the nose and pharynx, and not infrequently a serous fluid in the pericardial sac. In cases not quite so acute there will be found a catarrh also of the larynx and bronchi and frequently catarrhal pneumonia. The lungs in such cases are affected with red patches of lobular pneumonia, while a considerable area of the lung, particularly the lower and anterior portions, are dark red and more or less hepatized. On cutting these parts exudate can be pressed out. In older cases the pneumonia may be accompanied by pleurisy, the surface of the lung being dull and sometimes covered with a fibrinous exudate. In rare cases there is a quantity of fluid in the pleural sac. The mucous membrane of the mouth is not infrequently the seat of lesions ; the teeth become coated with a brownish catarrh offensive in odour, and ulcers may be observed on the gums, inside the cheeks and lips, and in rare occasions the tip of the tongue may be affected with necrosis and gangrene. When the lower part of the alimentary tract is involved one of the first areas to be affected in acute cases appears to be the ileo-cæcal valve, which in acute cases presents a crimson ring of congestion. If the post-mortem examination is delayed this lesion is not so readily observed and loses its brilliancy, becoming bluish-grey in colour. In the less acute cases the mucous membrane of the stomach or intestines, or both, is thickened and catarrhal, and often acutely inflamed, and occasionally shows superficial ulcers. The peritoneal cavity often contains a small amount of yellowish serous liquid. The liver and kidneys are congested and easily broken down, but these conditions are not characteristic of distemper. The lymphatic glands throughout the body are slightly enlarged and swollen and sometimes hæmorrhagic. In animals that have died with nervous complications, the membranes of the brain and spinal cord may show some congestion, and there may be a little fluid in the spaces. In other cases, however, there are no changes to be found in these structures visible to the naked eye.

Symptoms.—The symptoms of distemper usually commence with some slight depression, lassitude, and reduced appetite, together with a slight moisture from the nostrils. On taking the temperature it is found to be elevated 3° or 4° F. If the eyelids are everted, the conjunctiva is seen to be very watery. On examining the skin at this time slight small vesicles may be observed inside the flank and thighs, very quickly developing into flat pustules. Following this an occasional cough is heard and the nasal catarrh becomes thick and purulent and a similar discharge escapes from the eyes. The muzzle becomes hot and dry and sometimes cracked, while the discharge may be caked about the nostrils, which may be almost blocked, causing breathing through the mouth.

At this point various complications may arise involving either the respiratory tract, the alimentary tract, the nervous system or the skin, setting up what are known as the respiratory, the abdominal, the nervous and the cutaneous forms of distemper. Several of these forms may occur simultaneously.

The Respiratory Form.—In this form the primary distemper becomes complicated with bronchitis and pneumonia and sometimes pleurisy. Nasal catarrh extends to the trachea and bronchial tubes, and causes constant and pronounced coughing. The respiration becomes difficult, the head being extended and nostrils dilating and contracting during each respiration, and in advanced cases the cheeks also. When the pleura becomes involved there is pain over the chest wall, and if the dog is picked up it generally sets up a fit of coughing. The pain is relieved when exudation into the chest occurs, but the inspiratory effort becomes more exaggerated.

On auscultation a roughened vesicular murmur is first heard, together with moist râles (the sound of air passing through or over mucus in the bronchial tubes). This is followed by loss of sound due to consolidation of the part, particularly of the lower and anterior parts of the lung. With pleurisy friction sounds are observed in the earlier stages, while the movement of the fluid may be noted in the exudative stage.

In the respiratory form the respirations are increased to about 80 per minute, and in the advanced stages they are very shallow, and as the patient becomes weaker the pulse also becomes weak and feeble.

Lesions of the eyes frequently occur, along with the respiratory form of distemper. They usually commence with conjunctivitis and opacity of the cornea. This may be followed by the formation of an ulcer, particularly in the case of dogs with prominent eyes. There is photophobia (a condition in which light causes irritation of the eyes which are accordingly usually kept closed, the patient seeking dark corners of the room). In unfavourable cases the deeper layers of the cornea protrude through the ulcer, forming a staphyloma which may rupture and be followed by protrusion of the iris. Infection of the eyeball may then take place, setting up ophthalmia which may result in complete blindness. At other times the ulcer heals with the formation of a vascular area or pannus with a more or less permanent opacity.

The Abdominal Form.—The alimentary complications of distemper are usually first shown by considerable thirst and vomiting of a yellowish slimy material. The bowels are irregular, constipation in the early stages being followed by diarrhœa, which is sometimes blood-stained. When the lower part of the bowel is affected severe straining may be set up, even though the bowel may be empty, and owners are apt to consider the straining to be a sign of constipation, especially in the absence of fæces. The mouth and teeth become coated with an offensive brownish material which is easily wiped off. Ulcers develop along the gums and lips and occasionally on the tip of the tongue and inside the cheeks with a yellowish necrotic material firmly adherent. During this complication the loss of flesh is very pronounced, probably more so than in the pulmonary form of distemper.

The catarrh of the bowel may extend to the bile ducts and set up the so-called *biliary form* of distemper with acute jaundice. The mucous membranes suddenly become intensely yellow, the temperature rises rapidly and fits often occur. In this condition the treatment is urgent, or it is likely to prove fatal within twelve hours.

Cutaneous Symptoms.—The characteristic skin lesion of distemper consists of a pustule, flat on the surface, from $\frac{1}{4}$ in. to $\frac{1}{2}$ in. in diameter, and containing a creamy yellow pus. These cutaneous lesions may last one or more days, and are sometimes followed by successive crops throughout the course of the affection. When they occur on the back they may cause matting of the hair and irritation, as the result of which the dog may cause himself considerable injury. Sometimes the skin lesions disappear very quickly, and all that is left to mark their previous existence is a spot apparently cleaner than the surrounding skin and outlined with a dark rim of ragged epithelium. The cutaneous lesions of distemper may be regarded as a form of impetigo, but they are by no means constant. In the writer's experience they may be found in about 50 per cent of cases if carefully looked for. The occurrence of the cutaneous lesion has given rise to the view that distemper of the dog is a form of Variola or Dog-pox, but this is negatived by the fact that the lesions do not go through the characteristic stages of a true variola, by their rapid development, and also by the fact that they are by no means constant in occurrence.

An entirely different skin lesion is sometimes met with affecting the limbs in the later stages of distemper with nervous complications such as Chorea. It consists of a series of vesicles, rapidly developing into ulcers, running down the whole of the limb apparently following the course of the sciatic nerve and its branches. It is probably due to interference with trophism owing to the nervous affection, and may be regarded as a form of *herpes zoster* or *shingles* (see "Eczema"). The lesions are very irritating, and the dog frequently bites them severely, causing considerable self-mutilation. Cases have been recorded in which animals have actually bitten off their hind limbs, while at other times similar mutilation has arisen in the region of the flank, resulting in perforation of the abdomen. These skin lesions, differing from the pustules, are very resistant to treatment, and local applications beyond simple protectives are useless. The condition is only likely to improve coincidentally with a general improvement in the nervous system.

Nervous Form.—The nervous system may become deranged in a varying degree of intensity in distemper. In the acute stages there may be intermittent convulsions or acute encephalitis, while in the later stages there may develop paresis of either the hind or all four limbs, or chorea may be set up more or less as a sequel. These nervous complications are most frequently seen in dogs with a highly excitable temperament and in in-bred dogs. The convulsions of the earlier stages of distemper are sometimes observed before the catarrhal fever is recognized. The dog is rather strange in his manner and has alternate attacks of excitement and depression. The convulsions primarily affect the muscles of the head and neck. The face twitches, the lips are retracted and the teeth exposed and chattering, while the head is thrown back and the dog falls down. The seizure usually lasts less than a minute and may occur several times in the day or in rapid succession, leaving the animal very exhausted. Encephalitis as a complication, together with meningitis, usually occurs several days after the catarrhal symptoms sometimes not very pronounced have been observed. The patient becomes dull and unsteady on his limbs. He develops a staring expression, pupils are dilated and he may become quite blind, running into obstacles. He frequently moves forward until he arrives with his head in a corner and seems unable to extricate himself. This is followed by a twisting of the head in one direction or the other, and frequently paralysis of his limbs. It frequently happens that at this period the dog rolls over on the long axis of his body until he arrives at an obstacle beyond which he cannot pass. If he is propped up in this position he will frequently remain stationary and restful for hours. The patient will frequently take food quite readily if it is put into his mouth, or if he is held with his lips in contact with it. Such cases are frequently, but by no means invariably, fatal, much depending upon the nursing.

Paralysis of the limbs occasionally occurs during the later stage of distemper when the catarrhal symptoms are abating, and the patient appears to be well on the way to recovery. It usually commences with the hind limbs with a rolling gait, the limbs become weaker until completely paralysed. The dog drags himself along with his fore limbs, the hind limbs trailing behind. In many of such cases the appetite is well maintained and complete recovery follows. At other times the paralysis extends gradually forwards, involving the fore limbs and later the neck and head. Vomiting of nervous origin frequently follows, and the animal becomes rapidly weaker and almost invariably succumbs.

Chorea may develop in the later stages of distemper, and there appears to be no relationship between the severity of the attack of distemper and the liability to the onset of chorea. In fact it often occurs where the catarrhal symptoms have been so mild as to be overlooked, until the owner is cross-questioned as to the animal having been affected with a slight cough or catarrh a week or two previously. Chorea usually commences in one limb, most frequently a hind limb, or in the masticatory muscles, or it may be first observed by the twitching of an eyelid. It is sometimes followed by recovery, while at other times it is rapidly progressive, causing paralysis of the hind limbs and relaxation of the sphincters of the bladder and anus, and in such cases terminates fatally (see "Chorea").

Prognosis.—Prognosis of distemper should always be very guarded; it is apparently quite impossible to foretell the termination. It frequently happens that the cases which appear to be the mildest succumb, and on the other hand animals that appear to be worse often recover. Taken as a whole the mortality will range between 30 and 50 per cent. The death-rate is always greatest in dogs less than six months old, and is gradually reduced with advancing age. It is higher in the finely bred and pedigree dogs than in the coarsely bred dogs and mongrels. The mortality is higher in those breeds in which in-breeding is practised, and that applies especially to the various toy breeds where diminutive size is aimed at. The short-nosed breeds suffer very severely, and the Japanese spaniel particularly may be mentioned in this respect. Relapses sometimes occur, probably due to reinfection, and such cases usually terminate fatally. The mortality of distemper, however, varies from season to season. Probably this depends upon the prevailing type of distemper of the particular year, the malignant pulmonary and the acute nervous forms being the most serious.

Treatment.—In the treatment of distemper attention to general hygienic conditions and nursing must occupy the first place. The judicious selection of drugs, according to the type of disease, may assist towards recovery if they can be administered without distressing the patient. The affected dog should be isolated and placed in a room with an equable temperature, but well lighted and well ventilated. The best temperature of the room is from 50° to 55° F. The kennel or room should be kept clean and sweet by the liberal use of soap and water, and some disinfectant such as sanitas fluid or miscible pine oil. In cold weather the dog should have a woollen coat applied and secured in such a way that it does not come off when he changes his position. The patient should be allowed to rest as much as possible; there is no objection to him sleeping more or less the whole time during the intervals between feeding, etc. Exercise out of doors is contra-indicated. He should be provided with a bedding such as pine chips or straw or a blanket, which can be destroyed when soiled. His coat should be kept clean and sweet, and although a bath should not be given any soiled parts may be washed provided they are thoroughly dried at once. The rest of the coat should be groomed and kept clean, but as the process is rather exhausting to the dog it should not be at all protracted. The eyes and nose should be kept clean by sponging frequently during the day, while a mild eye lotion such as boracic acid, grs. x.-℥i., should be flooded into the conjunctival sac. The eyelids should be raised and gently manipulated in order to remove the catarrhal exudate which frequently accumulates, particularly under the membrana nictitans. The edges of the eyelids may then be smeared with a little boracic ointment or weak oxide of mercury ointment (gr. v.-℥i.), to prevent them becoming glued together with the discharge. The nostrils after being wiped should be similarly smeared with boracic ointment to prevent caking of the discharge and cracking of the muzzle. The mouth should be mopped out several times daily with a weak solution of permanganate of potash, and the teeth should be cleansed with lint. It is a matter of no importance if the patient should swallow the permanganate solution during the process—in fact, in the alimentary form it is probably advantageous.

The diet of dogs with distemper is of very great importance. There is no hard and fast rule that can be laid down as suitable for all cases, and food should be selected according to the individual case and the organs involved. In the majority of cases the animal refuses to take solid food voluntarily, but will frequently lap a small quantity of milk or milk and barley water. A little beef-tea is also excellent, while Brand's Essence of Beef, or other concentrated meat juice, will often be found serviceable. These may be taken voluntarily in small quantities; if not, they should be administered slowly and carefully in order not to set up coughing. In no case should any food or medicinal agents be administered by the mouth if the dog forcibly resists, since the exertion and fatigue thus produced are likely to do far more harm than the food or medicine will do good. The white of an uncooked egg beaten up with a little less than a teacupful of milk, to which has been added a dessertspoonful of brandy, makes one of the best foods, particularly in the abdominal form of the disease. This mixture should be administered in small quantities of about one dessertspoonful at a time every one and a half to two hours. Solid food of any kind is contra-indicated when the alimentary tract is involved. In the pulmonary and nervous forms, however, the patient will frequently take a little solid food more readily, and in that case they may be offered small quantities of steamed white fish, finely minced beef or horse-flesh raw or cooked, or a little boiled rabbit. In any case, however, foods should be given *little and often* —that is the secret of success. If there is marked thirst for water it should not be gratified since it frequently brings about vomiting, particularly in the alimentary form. Moreover, if the patient has the opportunity to drink water he is less likely to voluntarily take liquid nutritives.

The medicinal treatment of distemper must depend upon the particular case. In simple uncomplicated cases internal antiseptic and febrifuge treatment should be adopted with the addition of a little stimulant. Amongst the best agents for this purpose are quinine (gr. ½-v.), and the various salicylates such as aceto-salicylic acid, sodium salicylate, salol and salicylate of bismuth (gr. i.-x.). These may be administered easily in pill form without danger of setting up a fit of coughing. As a general stimulant the brandy already suggested with the milk, or a little aromatic spirits of ammonia, will suffice. If the medicine has to be given in the form of a draught the following will be found useful:

℞ Sod. salicyl. gr. ii.-v.
Tinct. scillae
Spirit. ammon. aromat. } āā mm. x.-xxx.
Aq. chloroformi q.s.
Ft. mistura.

This may be repeated three or four times a day. The general vitality of the patient may be assisted by the administration of nuclein (nucleinic acid 2½ per cent), which may be given hypodermically or by the mouth in doses of ʒss.-ʒii. or ʒiii. once daily.

In the respiratory form of the disease medicated inhalations with eucalyptus or terebene afford considerable relief in assisting to remove the copious nasal discharge. It is often advisable to apply counter-irritants to the chest wall such as a flannel wrung out in very hot water and sprinkled with a few drops of turpentine, or a little linimentum saponis B.P. may be well rubbed in, to be followed with a woollen coat. A mild mustard plaster is similarly useful in short-coated dogs. In this form expectorants are more or less necessary, such as squills, glyco-heroin, compound tincture of camphor or small doses of chlorodyne. Ipecacuanha either in the form of vinum ipecac., or pulv. ipecac. co. (Dover's powder) gr. iii.-x., often affords relief. Another useful combination consists of potass. iod. gr. i.-v., tinct. nucis vom. mm. i.-viii. and tinct. scillæ mm. x.-xxx., aq. destil., q.s. This may be administered twice daily.

In the abdominal form of distemper alimentary sedatives and disinfectants are necessary. Vomiting is best allayed by the use of carbonate or salicylate of bismuth gr. ii.-x. combined with chlorodyne mm. i.-v. and repeated three or four times daily. The administration of brandy in the milk as already suggested reduces vomiting and should be alternated with the above. If there is no diarrhœa calomel gr. ¼-i. may be given once daily for several successive days until a reasonably free action of the bowel is obtained. Calomel is one of the best alimentary disinfectants known. Salol gr. ii.-x. is also an excellent disinfectant and febrifuge. If diarrhœa occurs an attempt should be made to control it owing to its very debilitating effect if left unchecked. For this purpose the chlorodyne and bismuth mixture already referred to may be sufficient. If not, tannoform gr. ii.-x., tannate of bismuth or tannalbin may be given. In obstinate cases Gray claims successful results from the use of a mixture composed of infusion of cloves, log-wood and diluted sulphuric acid. It has been suggested that it is unwise to check the diarrhœa of distemper, but while agreeing that a soft or semi-liquid condition of the fæces calls for no special treatment the writer is of opinion that acute diarrhœa is very prejudicial to recovery if left unchecked. Moreover, the irritation of the bowel to which it is due sets up violent straining, and is frequently responsible for intussusception or prolapse of the bowel. Severe straining may be relieved by rectal injections of normal saline solution (sodium chloride gr. iv.- water ℥i.), or of permanganate of potash (1 in 2000).

When the mouth is the seat of ulcers and areas of superficial necrosis the yellowish patches should be snipped off with curved scissors and the ulcers thoroughly cleansed with eusol and finally painted with pure tincture of iodine; subsequently the lesions must be dressed frequently each day with permanganate of potash solution or eusol.

When the liver becomes complicated, setting up jaundice, the treatment should be energetic. An emetic should first be given such as apomorphine hypodermically to be followed by a dose of calomel, and subsequently by small doses of sulphate of soda, together with repeated small doses of brandy.

In the nervous form of distemper where there are fits or convulsions they should be controlled with bromide of sodium ammonium or potassium gr. v.-xxx. combined with syrup of chloral mm. x.-ʒi. An ice-pack may be applied to the back of the head. After the acute meningeal symptoms have passed away the sedatives may be replaced by potassium iodide and tincture of nux vomica, or Easton's syrup or arsenic (Fowler's or Donovan's Solutions) and compound syrup of phosphates. At the same time a blister of biniodide or mercury may be applied to the back of the head. In the less acute nervous complications the stimulant treatment may be adopted at the outset, and in the case of chorea or paralysis commencing with the hind limbs a biniodide blister should be applied over the loins. It has been suggested that lumbar puncture may be of service in these cases, but in the writer's view no value can be attached to them. The use of the galvanic battery may be resorted to in the less acute choreic stages, but its value has been grossly exaggerated, and the writer has only found it of service in very exceptional cases.

When the catarrh of the eyes is followed by ulceration of the cornea more energetic treatment is required. Whatever agents are used should be weak and mild, but should be applied *frequently*. Amongst the best agents are :

℞ Zinci sulph. gr. iii.
Acid borici gr. x.
Aq. destill. ℥i.
Ft. collyr.

If there is much irritation ext. belladonn. liq. mm. v. should be added to the above Sodium hyposulphite (gr. x.-℥i.), silver nitrate (gr. ii. ℥i.), collargol (1 to 5 per cent) are also useful in these eye cases. When opacity of the cornea occurs a little calomel may be blown into the eyes once or twice daily. When small blood-vessels develop across the cornea the weak eye lotions may be followed by adrenalin hydrochlor. (1 in 1000) mm. x., in 1 per cent collargol ℥i., applied by dropping several times a day.

The lesions of the cutaneous form do not call for any special treatment beyond simple cleanliness and sponging with any mild antiseptic, after which they may be dusted with pure boracic acid, starch or bismuth subgallate.

When the patient has recovered from the acute stages of distemper and has become convalescent the various febrifuges should be discontinued and should be replaced by a course of tonics such as cod-liver oil, extract of malt and compound syrup of phosphates, or vegetable tonics, such as infusion of calumba and gentian.

Prevention.—With regard to the prevention of distemper it must be borne in mind that the disease is solely due to contagion, and that no dog can contract distemper unless it has been in contact with an affected animal or its discharges which may have contaminated food or water, kennels, etc. Many laymen imagine that all dogs must of necessity have distemper at some period of their life, but although it is a fact that most dogs do contract the disease owing to its ubiquity, it is by no means necessary that all should contract it. The rational preventive measure, therefore, consists in avoiding exposure to infection, and in this connection all dogs affected with any catarrh, particularly of the upper respiratory tract, should be regarded as suspect. It is often argued that a catarrhal affection in a particular case cannot be distemper, because the dog has been known to have had the disease sometime, perhaps years, previously. That, however, is a fallacy since although recovery from one attack confers a considerable degree of immunity it is not uncommon for dogs to have a second attack of distemper, which is, however, usually milder than if not preceded by a previous attack. The mildness of this attack may disarm suspicion, but animals so affected should be rigidly isolated, or at any rate young susceptible dogs should not be allowed to come into contact with them or their discharges. The common practice of allowing dogs to run free in the street is perhaps the commonest method of spread of the disease, and consequently the soundest method of prevention consists in keeping them, and particularly all valuable dogs, under strict control when at exercise in the street. New dogs purchased or brought into the kennels should be quarantined for three or four weeks in order to ensure that they are not in the incubative stage of the disease and thereby introducing infection. The same applies to dogs that have been exhibited at shows or have been even for a short period of a few hours in a dogs' home or kennels where numerous dogs are admitted. No exception should be made even in the case of veterinary hospitals, where the rule obtains that no dogs suffering from distemper are admitted, for in such institutions, at sometime or other, in spite of every possible care, a dog will be admitted either in the incubative stage of the affection or affected with the disease in some atypical form which could not be recognized. On the other hand, it should be borne in mind that if a dog should develop symptoms of distemper within two or three weeks after admission to a kennels or veterinary hospital, it cannot be taken for granted that he contracted the disease there, for the period of incubation not infrequently exceeds that period and infection may therefore have taken place before admission. Similarly, if a dog should develop symptoms within even a week or so after having been in such a place it cannot be positively assumed that infection occurred there, since in many cases of acute distemper the period of incubation may be as short as three or four days, and infection may have taken place by some means subsequently.

Immunization.—Numerous vaccines and sera have been introduced for the prevention of distemper, but in the opinion of the writer there is no immunizing agent yet produced which can be relied upon. It is possible, however, that some of the vaccines prepared from organisms which have frequently been found in dogs affected with distemper and are probably responsible for secondary lesions may exert some beneficial effect in reducing complications during the natural course of distemper. In the writer's experience, however, none of them produce any immunity against the actual disease itself. In this category may be included the vaccine prepared by Ferry and others from various strains of bacillus bronchi-septicus canis, which may reduce pulmonary complications; also vaccines prepared from various strains of *Bacillus coli* obtained from dogs affected with the intestinal form of distemper, and which may reduce the liability of gastro-enteritis. On the other hand numerous agents have been placed upon the market which are absolutely worthless as preventives to distemper and some have actually proved themselves dangerous. In those circles where distemper of the dog is regarded as a form of variola true vaccination with calf-lymph has been recommended. In the view of the writer such vaccination is worthless, but as it is also harmless there is no great objection to its use in those cases where owners insist on some form of vaccination. The inoculation usually takes place inside the ear-flap or inside the thigh.

G. H. W.

DISTEMPER OF THE CAT

Catarrhal Fever of the Cat ; Feline Influenza

The cat is the subject of a disease somewhat corresponding with distemper of the dog but with apparently no etiological connection. As in the case of the dog it is probably due to a filtrable virus, but little or nothing is actually known of its etiology. The writer has frequently seen instances of young cats being in constant contact with dogs affected with acute distemper, even to sharing their beds and licking their faces and discharges without themselves becoming in any way affected. On the other hand, he has also occasionally met with catarrh in the cat occurring simultaneously with distemper in the dog, but in view of the former experiences he has regarded such cases as pure coincidence. Conversely the writer also has experience of acute outbreaks of distemper in cats occurring in infirmary kennels also occupied by puppies of susceptible age, and although the latter had not previously been affected with distemper, in no single instance did they contract it from the cats.

Outbreaks of contagious catarrh of the cat have been described from time to time under the name of Feline Influenza, under the impression that they differ essentially from feline distemper, but a careful consideration of the symptoms recorded in such outbreaks has convinced the writer that they were actually outbreaks of feline distemper.

Infection.—As in the case of distemper of the dog the affected cat is the chief method of spread of feline distemper. This may occur when cats are allowed their liberty and also in institutions where cats with various affections are admitted for treatment, livery or sale. Recovery from one attack usually leaves some degree of immunity, but it is by no means absolute, and there are numerous instances of cats being affected two or three times.

As in the case of dogs distemper may occur in cats of all ages, but kittens under six months old are most susceptible.

The *period of incubation* is from three days to three weeks, and the course of the disease averages from seven to ten days, though in many cases it may extend to several weeks.

Symptoms.—Distemper of the cat assumes different forms from time to time. In some outbreaks the lesions are limited to the mouth and throat. At other times the pulmonary form is met with, and at other times abdominal form of distemper occurs. In each case there is a preliminary stage of fever, the temperature rising to 105° to 106° F., and slight catarrh, usually thin and watery, of the eyes and nose. Occasionally the nasal discharge is thick and yellowish-white, but this discharge is rarely so thick and tenacious as in the case of canine distemper. It is usually associated with sneezing and occasional fits of coughing. The appetite is variable, and in those cases where it is in abeyance for several days there is marked loss of condition. This is most pronounced in the abdominal form of the disease, which is generally the most acute form.

The mouth lesions of feline distemper usually consist of small vesicles or blisters along the margin and the tip of the tongue, and also on the roof of the mouth and inside the cheeks. The blisters rupture, and red angry-looking ulcers are produced which are very sore and completely prevents a cat from taking any food, solid or liquid, although it may be very thirsty, and will sit with its head over water or milk, evidently anxious but afraid to take it. In such cases if milk or even finely minced solid food is placed in the mouth the cat will often swallow it quite readily and look for more. This condition is usually associated with dribbling of thick tenacious saliva, and constipation which can be readily felt on palpation through the abdominal wall. This form usually runs a mild course if put under suitable treatment, otherwise it may become protracted and terminate fatally from emaciation and exhaustion. When the pharynx is affected similar lesions may be observed at the back of the tongue, and sometimes associated with a yellow fibrinous (diphtheritic) deposit. This form is more severe than when the mouth alone is affected, and produces difficulty of swallowing and frequent coughing. Manipulation of the throat from the exterior produces gulping, and is sometimes more or less painful. When the epiglottis is involved laryngitis and bronchitis frequently supervene with fatal results.

In the pulmonary form of the disease, which is much less common than the buccal form, the primary symptoms commence in a similar manner with fever, an occasional cough and constipation. Broncho-pneumonia develops rapidly with quick and difficult breathing and complete loss of appetite. Pleurisy sometimes supervenes, making respiration still more difficult and causing the cat to cry out in pain when lifted up. Emaciation takes place rapidly, and this form is very frequently fatal.

The abdominal form of distemper in a cat is also comparatively rare. It runs through a rapid course and is probably the most fatal form of distemper in the cat, death often occurring within eighteen or twenty-four hours after the onset of symptoms. In other cases it is less rapid and the course may extend over several days or a week, and even then is usually fatal. The symptoms consist of catarrh of the eyes and nose, sudden loss of appetite and vomiting, the vomit being sometimes frothy, at other times viscid and yellowish-green in colour. Diarrhœa sets in within a few hours. It is extremely offensive in odour and often blood-stained. The cat rapidly becomes weak, its extremities very cold, cries out if manipulated, becomes comatose and dies. In less acute cases the course is less rapid and the exhaustion and emaciation more gradual. If the skin is raised in a fold it is slow to resume its normal position. Mouth lesions are also frequently met with in these cases, but the ulcers are generally very offensive, thus differing from the ulcers in the mild form of the disease.

Skin eruptions and nervous complications have been recorded in feline distemper, but they are extremely rare, and in a very extensive experience of the disease the writer has rarely met with either.

The *mortality* of the disease varies considerably with the type of outbreak and the breed of cat affected. In the simple form the disease is rarely fatal, but in the pulmonary form the death-rate is from 50 per cent to 75 per cent, while in the gastro-enteritic form the mortality may be up to 90 per cent. It is more severe in Persians, Chinchillas and Siamese cats than in the general utility cat.

Treatment.—The affected cat should be immediately isolated and placed in a room with the best possible hygienic conditions as to light, ventilation, warmth, etc. Its strength should be maintained by judicious feeding with milk and white of egg, which should be administered with a spoon if not taken voluntarily. Brand's Essence and meat extracts may be similarly given except in the gastro-intestinal cases, when they are best avoided; Benger's food, lactol and arrowroot being substituted.

Medicinal treatment varies according to the case. In the simple cases with the primary fever and constipation a single dose of a mild aperient should be given, such as calomel gr. i. in pill. The ulcers of the mouth should be treated with simple mouth washes, probably the best being boracic acid solution gr. x.-ʒi., a teaspoonful of which should be placed in the mouth at frequent intervals during the day. The fever should be combated with quinine pills gr. i. or aceto-salicylic acid gr. ii., twice daily. The same medicinal measures may be adopted in the pulmonary form of the disease, but the addition of syrupus scillæ mm. x.-xxx. and glyco-heroin mm. vii.-x. two or three times daily will be found very useful as a cardiac and pulmonary stimulant. In cold weather a woollen coat or a bandage should be applied to the body or the chest. Counter-irritants in the form of liniments, etc., are contra-indicated owing to the presence of the fur which they leave wet and may thus be productive of more harm than good. In this form medicated inhalations assist the removal of catarrhal exudates. The eyes and nose should be kept sponged at frequent intervals with boracic solution.

In the gastro-intestinal form the disease is often so rapidly fatal that no medicines are of any service. In the less acute cases, however, carbonate of bismuth and tannoform āā gr. i.-iii. should be given three times daily, or alternatively bismuth carbonate gr. i.-iii. with chlorodyne mm. i.-iii. In some cases with persistent vomiting chloretone gr. i.-ii. is effective.

In each form of distemper of the cat the hypodermic injection of nuclein c.c. i. stimulates the activity of the white blood corpuscles and may be given with advantage once every day or on alternate days.

During convalescence a small amount of solid food should be offered, such as shredded steamed white fish or a little scraped or finely minced raw lean meat. If the cat is allowed to eat much solid food vomition is likely to be set up. At this time also with advantage may be given an emulsion consisting of syrup. ferri phos. co. ℥ss , ext. malti liq. ʒi.ss., Ol. morrhuæ up to ℥iv., one large teaspoonful twice a day mixed with an equal quantity of milk to reduce the tendency to cause vomiting.

If a case of feline distemper crops up in an infirmary or kennels all the other cats should be immediately sent home owing to the very highly contagious character of the disease. Very thorough disinfection should be practised, and several weeks or even a month should be allowed to elapse before cats are readmitted to the premises.

G. H. W.

CONTAGIOUS BRONCHO-PNEUMONIA IN DOGS

This is a contagious affection of the dog most frequently affecting adults and characterized by inflammation of the lungs and bronchi. It is very frequently diagnosed as distemper, but it appears to the writer to present various manifestations which are sufficient to justify it being regarded as an entirely different disease. In the first place it attacks old rather than young dogs, and with much more serious effects. Dogs known to have recovered from distemper and to have a considerable degree of resistance to that disease are readily infected with contagious broncho-pneumonia if brought into contact with affected animals, and may die in a few days. The period of incubation in the average case is very much shorter than that of distemper, and the disease appears to be limited to the respiratory tract. There are never any cutaneous lesions nor gastro-intestinal complications. Moreover, the writer has never observed any nervous complications or sequels such as chorea. The disease appears to be less widespread than distemper, but, like that disease, it is mainly spread by infected dogs and particularly by dogs' homes. In one large dogs' home known to the writer a stay of a few hours on the part of an adult dog is absolutely certain to infect him with this disease, which develops two or three days later.

Symptoms.—The symptoms develop with great dulness which is rapidly progressive, blowing respirations and frequent coughing—the temperature 105° to 106° F. The dog is almost completely off its food, but will often pick a little dainty morsel offered by hand. In a few days a discharge escapes from the eyes and nose, which is extremely viscid and has a great tendency to cake round the eye-lids and nostrils almost like glue. This discharge markedly differs from the thick purulent discharge usually encountered in distemper. On wiping away the discharge from the eyes the conjunctiva is seen to be brightly injected. There is also a great tendency to cracking of the skin of the muzzle. In some cases acute pleurisy develops with a painful condition of the chest wall. This causes the dog to cry out when picked up. A pleural exudate is soon thrown out and the painful stage declines, but the respiration becomes more difficult.

On auscultation in the early stages increased bronchial sounds are heard in patches, and areas of consolidation with loss of respiratory sound develop in a short time. The friction sound of pleurisy is observable in some cases, followed by loss of respiratory sounds during the exudative stage.

Treatment.—The affected animal must be rigidly isolated and provided with good hygienic conditions with plenty of warmth and plenty of fresh air. Attention should be paid to the diet, which should be concentrated and easily digested. Dogs with this affection will usually feed a little if coaxed. This is a great advantage since it obviates the dangers of artificial feeding. Medicated inhalations afford considerable relief. Care must also be paid to the eyes and nose to see that the discharge does not cake in either situation, and after its removal the edges of the eyelids should be smeared with a little boracic ointment. Medicinally, similar measures may be taken as are recommended for the pulmonary form of distemper, reliance being placed on internal antiseptics and febrifuges such as aceto-salicylic acid, quinine, potassium iodide, and also on expectorants such as tinct. camph. co., tinct. scillæ, glyco-heroin, vinum ipecac., and pulv. ipecac. co. Nuclein may also be given hypodermically or by the mouth. A long convalescence is necessary, during which the patient should have a course of cod-liver oil and malt and phosphate of iron. Very thorough disinfection of the kennels must be carried out subsequently.

G. H. W.

CANINE TYPHUS

Epizoötic Gangrenous Stomatitis and Gastro-Enteritis ; Stuttgart Dog Disease

Canine typhus is a disease of dogs characterized by hæmorrhagic gastro-enteritis with a great tendency to necrosis of the mucous membrane of the mouth and other parts of the alimentary tract. As in the case of contagious broncho-pneumonia in dogs this disease has frequently been regarded as a form of canine distemper, but there are good reasons for considering it to be an entirely different affection, one of the most important ones being that although it may attack dogs of all ages, it most frequently affects old dogs and is much more serious in them than in young dogs, being the converse of distemper in that respect.

The actual cause of canine typhus is not definitely known, but the lesions of the stomach and intestines usually contain an enormous number of organisms of the *Bac. coli* type which may, however, be due to a secondary invasion. On the other hand, *Bacillus coli* may possibly be the cause of the affection, and this view is somewhat supported by the results which frequently follow treatment of the disease by means of vaccines prepared from a canine strain of that organism.

The method of infection appears to be somewhat curious. Although the disease may occur in the form of an epizoötic or enzoötic with a considerable number of dogs in the locality becoming affected, one may meet with isolated cases with no history of contagion and yet showing exactly similar symptoms and lesions. Moreover, it not infrequently happens that a healthy dog may live with an affected one and still not contract the disease.

Etiology.—The experimental feeding of healthy dogs with discharges from affected animals or with portions of stomach or intestines has frequently failed to produce the disease, although there are recorded cases where canine typhus followed such procedure.

In some outbreaks the writer suspected that it was transmitted by dog lice or fleas acting as carriers, possibly mechanically. Organisms corresponding to those found in the lesions of the alimentary tract have been demonstrated in such insects taken from affected dogs.

Symptoms.—The symptoms of this disease vary considerably in their intensity. When it is newly introduced into a district it runs a very acute and rapid course. When it has been in existence for some time the majority of cases run a sub-acute course, while still later a mild or chronic form of the disease obtains.

The Acute Form.—In this form the disease runs a very rapid course and frequently proves fatal within twenty-four hours, although it may extend over several days. It usually commences with acute vomiting and great dulness and depression. At first the vomit is greenish in colour but rapidly becomes blood-stained and very offensive in odour. In the early stages the patient may take a little food and is very thirsty for cold water, but vomits immediately solids or liquids are taken. The fæces are at first firm with a tendency to constipation, but this is quickly followed by acute diarrhœa, the fæces containing a large quantity of blood and being extremely offensive in odour. The patient becomes very weak and loses flesh with remarkable rapidity. The abdomen is tucked up, the back arched and the dog looks very dejected and miserable; the eyes become dull and sunken. The mouth is cold and clammy, and a catarrhal coating is deposited on the teeth, dark brown in colour, becoming blood-stained and extremely offensive; there is frequently dribbling of cold saliva. By this time the animal is unable to stand, and the temperature, which was elevated several degrees at the outset, becomes subnormal. In those cases that last several days extensive ulceration of the mucous membrane of the mouth, particularly along the gums and inside the cheeks, and sometimes at the tip of the tongue, is set up, the patient becomes very prostrate, the extremities very cold, and semi-coma and coma supervene a few hours before death.

This form is fatal in from 80 to 90 per cent of cases, and although it may occur in any breed, in the writer's experience it has been more frequent and more severe in bull-dogs, especially those with a previous history of chronic gastric catarrh.

The Sub-Acute Form.—In the sub-acute form of the disease usually the first symptom observed is a cough, and on examining the mouth and throat a certain amount of congestion of the pharynx is observed. The dog may vomit once or twice after food, and yet is not suspected of being very ill. He is then noticed to become somewhat dull; the appetite may remain for a day or two, but the vomition becomes more frequent, the vomit being greenish or yellowish-green in colour. There is a tendency to drink a considerable quantity of water, after which the dog is likely to vomit. He may cry out if picked up as from abdominal pain. There is a slight discharge from the eyes which soon become dull and sunken. For the first day or two there is some constipation, followed by diarrhœa, which is subsequently blood-stained and very offensive in odour. Irritation of the lower part of the bowel causes constant straining (tenesmus), which is often mistaken as the sign of constipation. By this time there is a dribbling of saliva which is sometimes blood-stained; the mouth and lips are cold and clammy; necrosis of the gums and inside the cheeks is observed in the form of yellowish, firmly adherent shreds, which become detached, leaving angry-looking ulcers. The tip of the tongue becomes slightly congested and then brown, and this is followed by necrosis, which is indicated by the tip or edge of the tongue becoming yellow. This portion may fall off and in some cases the dog may actually lose the entire free portion of the tongue. The vomit may now be blood-stained and sometimes consists entirely of blood, and the vomiting spasms occur without food or water. The pulse becomes fast but weak; the temperature is at first elevated 2° or 3° F., but fever is never well marked; the temperature becomes subnormal as the disease advances, the extremities and body surface being cold. There are no visible signs of lung disease. By this time the patient is extremely weak and unable to stand, and presents a very dejected appearance. Emaciation becomes very pronounced, and if a fold of skin of the neck or back be raised it is slow to resume its normal position. Death is usually preceded by coma and collapse.

The course of the sub-acute form of the disease may extend from two to ten days and is fatal in at least 50 per cent of cases. The case is more hopeful when lesions and symptoms are limited to either the mouth or stomach or intestines, and especially so when vomiting and thirst are not very pronounced, as in those cases the patient's strength is more easily maintained.

In the *mild form* of this disease the symptoms more closely resemble those of simple gastric catarrh with frequent vomiting and great thirst. The patient is dull and takes very little food, the temperature may be normal or elevated 1° or 2°. In a few days coating of the teeth and ulceration of the mouth may occur, but in this form there is rarely necrosis of the tip of the tongue. The bowels are irregular, with a tendency to slight diarrhœa. Loss of condition becomes very pronounced. The condition described elsewhere as Contagious Ulcerative Stomatitis is often regarded as a mild form of this disease, but as it is rarely accompanied by vomiting or diarrhœa it is probably a different affection.

A *chronic* or atypical form of canine typhus is met with occasionally. The symptoms consist of persistent vomiting of a yellowish or greenish-yellow viscid substance, irregular bowels, great thirst, and progressive emaciation. Blood-staining of the discharges may be absent throughout the course. The mucous membranes are anæmic and the temperature is normal, or in the advanced stages, often slightly subnormal. Occasionally, when the vomit has been in existence for ten days or a fortnight, mouth lesions may be developed resembling those in the acute form of the disease. Such cases are usually fatal. In other instances the diagnosis of this condition is somewhat difficult, as the symptoms closely resemble those caused by obstruction in the intestines. The writer has on several occasions recommended an exploratory laparotomy, and although no foreign body was discoverable the results were satisfactory, inasmuch as vomiting subsequently ceased.

Anatomical Changes—Acute and Sub-Acute Forms.—The lesions are mainly confined to the alimentary tract. In the acute form they consist of ulcers of the mouth, brown coating of the teeth, frequently sloughing of areas of the gums and the free portion of the tongue. The stomach is devoid of food materials and often contains a quantity of clotted blood, very dark in colour and offensive in odour. The mucous membrane is inflamed, hæmorrhagic, and often ulcerated. The bowels vary considerably; in some cases they are inflamed and hæmorrhagic, particularly the small intestines, while at other times there is very little enteritis. The abdominal lymphatic glands are swollen, juicy, and often hæmorrhagic. There is usually great lack of fat in the carcase unless death has been very rapid.

In the chronic form of the disease it is a common thing to find no lesions whatever. At other times ulceration of the buccal mucous membrane is present.

Treatment.—Isolate the patient as soon as possible, putting him in a quiet place with good ventilation and a temperature of 55° to 60° F. Good nursing is of the greatest importance. The patient's strength must be maintained by the frequent administration of small quantities of milk and white of egg, giving at a time not more than from one to four dessertspoonfuls, according to the size of the dog. Its retention is facilitated by the addition of brandy, from twenty drops to a teaspoonful. Other milk foods may be substituted, such as glaxo, lactol, or Benger's food. Meat extracts are excellent in chronic cases, but are not so useful when the hæmorrhagic gastritis is present. Drinking-water should be completely withheld. The mouth must be thoroughly cleansed. At the first dressing all areas of necrosis must be removed and the parts scraped with a curette and disinfected with tincture of iodine or carbolic acid or strong solution of permanganate of potassium, sodium, or calcium. Subsequently the mouth must be mopped out and thoroughly cleansed very frequently during the day with weak solutions of the same agents, or with hydrogen-peroxide or saturated solution of boracic acid. The stomach may be irrigated by means of a stomach pump with permanganate solution (1 in 2000) or with normal saline solutions (sodium chloride gr. iv.-℥i.). When hæmorrhagic enteritis is present rectal injections of normal saline or permanganate appear to relieve the straining, and assuage the thirst at the same time.

Medicinally, in the early stages the symptoms may be relieved by the use of carbonate of bismuth, sodium salicylate āā gr. ii.-x., or bismuth salicylate or salol. These should be administered three or four times daily alternated with brandy in milk. Good results may sometimes be obtained by the administration of permanganate of calcium, sodium, or potassium gr. $\frac{1}{8}$-$\frac{1}{2}$ in pill. Other agents used include chinosol gr. i.-v., beta naphthol, sodium sulphocarbolate, boracic acid, and hydrocyanic acid. It often happens that everything administered by the mouth is speedily vomited. In such cases brandy or sulphuric ether may be administered hypodermically, and food must be administered in the form of nutrient suppositories. Hypodermic injection of nuclein will also be found serviceable.

In convalescence tincture of nux vomica, tincture of squills, and aromatic spirits of ammonia may be given in milk, and also cod-liver oil and malt. Water should still be withheld, and feeding with solids should only be resumed after several weeks, commencing with a little boiled sheep's paunch or a little scraped or finely minced meat.

Vaccine Therapy.—The writer has on numerous occasions resorted to the use of a serum and also a vaccine prepared from *Bacillus coli* (canine strain) with apparent good results. It may be said that this organism is not the specific cause of the affection, but if it is responsible wholly or in part for the lesions, a vaccine or a serum prepared from it may reasonably be expected to reduce the severity of the lesions and to contribute towards recovery. The writer's experience supports that contention. G. H. W.

CHRONIC METRITIS IN THE BITCH

A form of chronic metritis of bacterial origin occurs in the bitch and occasionally in the female cat. It is most commonly seen in aged bitches, in the majority of cases about eight years old, that have never been in whelp, though it is met with occasionally in young bitches and sometimes in aged bitches that have bred at one time but not for some years. All breeds of dogs appear to be equally susceptible.

Etiology.—The condition is almost always associated with the presence in the uterus of a small bacillus of the colon type, often in pure culture, and judging by the almost constant presence of this organism it is probable that it is the actual cause of the disease. In rare cases the organism apparently responsible for the condition is a streptococcus which is found in almost pure culture in those cases. From the fact that the disease is much more common than formerly and that a number of cases may be met with almost simultaneously one is probably justified in assuming that it is contagious, but the method of spread is unknown. The common method is certainly not by coitus, since it commonly occurs in virgin bitches whose history absolutely precludes service, promiscuous or otherwise. It is more probable that infection takes place by the mouth.

Symptoms.—Symptoms are usually first observed a week or two after the bitch has been in season. Frequently the first thing noticed is occasional vomiting, perhaps only several times a week, as the result of which a mistaken diagnosis of gastric catarrh or indigestion is often made. The appetite is usually good in the early stages but later becomes capricious. The temperature is rarely elevated, but may become subnormal in the advanced stages. The mucous membranes are usually pallid—occasionally they are slightly injected. The abdomen is observed to become more distended than normal, the distension being more marked along the lower third of the abdomen, usually bilateral, and giving in bad cases the appearance of an advanced stage of pregnancy. There may be some discomfort on palpation of the abdomen, but the trill observed on percussion of the abdomen in dropsy is absent. On examining the vagina a discharge may usually be observed, either leaden-grey or chocolate brown in colour, and very viscid. It may be present in varying quantities, and is sometimes only noticeable by the fact that the bitch leaves a moist spot when she sits. It sometimes happens that the vaginal discharge is slight but more or less continuous from the outset, and in such cases the abdominal distension may be absent. In prolonged cases the patient becomes emaciated and may succumb to exhaustion.

Lesions.—The uterus is found to be distended to a varying degree, and in some instances it may attain the size of one's forearm. When it is open it is found to contain a varying quantity of purulent material, either leaden-grey, yellowish-grey, or chocolate brown in colour. The uterine mucous membrane varies in appearance. In the majority of cases it is quite pallid, almost white, but occasionally it may be intensely congested and hæmorrhagic. The surface is generally coarsely roughened and may present villus-like projections. In other cases it is corrugated or thrown into folds. Sometimes it is quite smooth; at other times the mucous membrane shows innumerable small cysts which may extend from the Fallopian tubes along each horn to the body of the uterus. The cysts vary from the size of a lentil up to a haricot bean, and their contents may be either

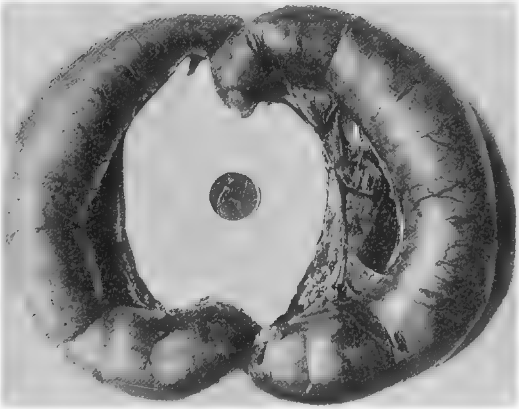
Fig. 32.—Uterus of bitch with chronic metritis, successfully operated upon. (The size may be estimated by comparison with the penny-piece.)

a clear serous liquid or hæmorrhagic. The ovaries are sometimes normal, but in the majority of cases they are cystic; the cysts may be as large as walnuts.

Treatment.—The most satisfactory treatment is undoubtedly excision of the womb, and the earlier it is done the better, as the recuperative powers are then quite good, and the patient generally makes a rapid recovery from the operation. If the condition is allowed to go on too long, however, the patient becomes a very bad subject for so severe an operation. If the owner objects, however, to surgical measures medicinal treatment may be resorted to and is sometimes effective. The writer has had good results in a number of cases from the administration of sodium salicylate and hexamin āā gr. iii.-x. twice daily. At the same time the womb should be irrigated daily with a mild antiseptic such as eusol or permanganate of potash, 1 in 4000. In a number of cases also autogenous vaccines have produced excellent results, while the writer has also been able to attribute good results to the use of a polyvalent stock vaccine of *B. coli* (canine strain).

G. H. W.

DISEASES DUE TO PROTOZOAN PARASITES

The *protozoa* are the lowest types of animal life. They are each composed of a single cell only, a point which differentiates them from all other animals which are formed out of a more or less considerable number of cells arranged in a special manner so as to perform special functions and are thus designated *metazoa*. The protozoa have to be distinguished again from other unicellular organisms such as the bacteria, spirochætes and a large number of plants (*e.g.* Algae). The composition and structure of the protozoan cell body exhibit the greatest similarity with the cells composing the tissues of the higher animals, while they are different from the lowest plants in that they contain no chlorophyll, and are thus unable to utilize simple inorganic matter as their nutriment. The bacteria resemble in their requirements and structure the lowest types of moulds. The spirochætes form a group which possess a considerable number of points of similarity with the protozoa (flagellates), but their structure appears to correspond somewhat with that of the bacteria; hence this group is sometimes allocated a position midway between the unicellular animals and plants.

Like all other animal cells the protozoa are composed of a more or less fluid living albuminous substance—protoplasm—which in all cases is differentiated into a cytoplasm, usually forming the bulk of the cell body and containing within it a highly differentiated more or less compact mass known as the nucleus, which is composed largely of a substance known as chromatin (on account of certain peculiar staining properties) and which appears to preside over the functions of the cell. But although all protozoan elements are essentially composed of these two parts only—nucleus and cytoplasm—they exhibit among themselves a very considerable degree of variety in shape and form, and parts of their substance may become highly differentiated (organelles) so as to perform functions analogous to those of the organs of the higher animals. The great majority of the protozoa are free-living and are found on the surface of the earth in salt- or fresh-water, especially stagnant water, and in the moisture of the soil, and they play a large part in the economy of nature. A large number live normally in the digestive tracts of animals, especially herbivorous animals, and their presence appears to exert no harmful effect upon their hosts, and in some cases they may even render some aid in the digestive processes; these are what are known as saprophytes. In comparatively few instances, however, the parasitism of certain of these protozoa has a most harmful effect upon the host, and thus some of the most widespread diseases of both man and the lower animals may be put to the count of such organisms. Examples are malaria in man, red-water or Texas fever in cattle, and the formidable diseases known as nagana and surra among stock in Africa and India respectively. Many of these protozoa exhibit highly complex life cycles.

The sub-kingdom Protozoa has been variously classified, one of the most recent classifications (Doflein) being the following:

Group 1. Plasmodroma.—Protozoa possessing flagella or pseudopodia or having no special locomotory appendages; one or more, usually vesicular, nuclei, which nucleus is never differentiated into a nucleus and paranucleus. Multiplication always takes place as the result of copulation, that is, the union of male and female elements, although this process may only take place at rare intervals during the multiplication, and the forms exhibited by the organism before and after copulation usually show considerable differences. This sub-family is divided into three classes: Class (*a*) Mastigophora, which move about by means of whip-like processes (flagella); Class (*b*) Rhizopoda, which move about by throwing out protoplasmic processes (pseudopodia); Class (*c*) Sporozoa. These have no locomotory appendages or pseudopodia, they are always parasitic. After copulation they multiply by dividing into spores, which are usually numerous and in most cases furnished with a protective covering.

Group 2. Ciliophora (Infusoria).—These move about by means of vibrating hair-like processes (cilia) and they contain a large nucleus and a vesicular paranucleus. They exhibit no alternation of generations. Division takes place by the same method both before and after conjugation, viz. by simple fission or by budding. This group contains two classes, viz. (*a*) Ciliata, and (*b*) Suctoria. These, however, are all free-living organisms or saprophytes.

CLASS MASTIGOPHORA

This class of flagellate organisms has been divided into a large number of orders, families, and genera, but the most important from the disease-producing point of view are the representatives of the trypanosome family—Trypanosomidæ — containing the orders Leptomonas, Herpetomonas, Trypanosoma, Schizotrypanum, Endotrypanum, Leishmania.

The more important trypanosomiases—including the " African trypanosomiases," surra, dourine, and mal de caderas—are specially dealt with by various authors (*q.v.*). J. T. E.

TRYPANOSOMIASIS

Trypanosomiasis as a disease of domesticated stock first acquired prominence in 1880, when Griffiths - Evans, an Inspecting Veterinary Officer of the Northern Command in India, discovered that the disease there known to the natives as surra was due to a trypanosome.

In 1894 Sir David (then Major) Bruce, R.A.M.C., investigated a disease of domestic stock in Zululand, locally called *nagana*. Bruce showed that this condition was due to an infection with a trypanosome conveyed by the bite of the tsetse fly (*Glossina morsitans* and *Glossina pallidipes*); and he further demonstrated that wild game living in that part of Zululand harboured trypanosomes in their blood, and were consequently to be regarded as the reservoirs from which the tsetse derived infection.

In 1901 Ellmassian described a form of trypanosomiasis under the name of mal de caderas, occurring near the mouth of the River Amazon, and in parts of Bolivia and Paraguay. This disease principally infects horses, to which infection is believed to be transmitted through the bites of the stable-fly (*Stomoxys calcitrans*), and the reservoir of the virus, equivalent to wild game of Bruce's *nagana*, is considered to be the rodent *capybara*.

In the same year Dutton described the first case of trypanosome infection of the human being. The infection was recognized in the blood of a European on the West Coast of Africa, and when the disease known as sleeping sickness—at that time decimating Uganda—was described in 1903 it was found that Dutton's trypanosome (*Trypanosoma gambiense*) was the causal agent.

Since that date over twenty-five species of *Trypanosoma* have been isolated from or described in the blood of domesticated animals, the majority occurring on the continent of Africa.

It had been known since the discovery by Gruby in 1843 that protozoa of the genus *Trypanosoma* existed in the blood of other animals. Gruby's discovery was made in the blood of a frog, but Valentin two years previously had seen similar bodies in the blood of a trout.

The first discovery of trypanosomes in mammals was made in India in the year 1878 by Lewis, who detected them in a very great number of rats there examined.

The presence of trypanosomes, of which there is a large number of species, in the blood of reptiles, amphibians, birds, and small mammals is not to be regarded as necessarily detrimental to the host. *Trypanosoma lewisi*, for example, the parasite discovered in rats by Lewis, has been found to have an almost world-wide distribution, to be met with in rats of almost every age, and rarely to produce any symptoms in the host animal. Most of these parasites are therefore regarded as non-pathogenic.

Among the trypanosomes of domesticated animals is a species (*Trypanosoma theileri*) first discovered by Theiler in South Africa in 1903, and since that time found to occur in Europe, Asia, and America, which is now regarded as being comparatively harmless to its host. By means of very large injections of heavily infested blood it has been possible to induce mild febrile symptoms of an ephemeral character associated with slight anæmia and jaundice in young bovines and in others possessing a peculiar degree of susceptibility.

The forms of trypanosomiasis with which we shall deal in this article are those in which the *Trypanosomata* invade the peripheral circulation, and by their presence give rise to febrile reactions, to anæmia of progressive character; and they are in nature transmitted from host to host through the agency of a biting fly.

There is, however, one form which causes the disease known as dourine that does not invade the peripheral circulation to the same degree, and which is spread from animal to animal by coitus.

Economic Importance.—In tropical Africa there is no disease causing more loss to stock-owners, or which more entirely precludes the keeping of domestic animals over enormous tracts of territory otherwise well suited for stock grazing, than trypanosomiasis.

In Africa, Northern Rhodesia, Nyasaland, Portuguese East and Portuguese West Africa may be taken as the southern limits of the badly infected countries, although infection still exists in more isolated areas as far south as Zululand. To the north the disease extends to the Sudan and French West Africa in an epizoötic form; while in Egypt, Tunis, and Morocco outbreaks not infrequently occur.

The Aden hinterland, southern coast of Arabia, the littoral of the Persian Gulf; Baluchistan, India and Burma, the Malay States, French Indo-China, and the Philippine Islands, all suffer from periodical outbreaks of surra.

In South America mal de caderas is assuming a wider distribution each year, and it is now being recognized in States hitherto regarded as free.

The ravages of sleeping sickness in Uganda, and round the Great Lakes of Central Africa, and on the big rivers of the Congo Basin, are still fresh in the public mind.

Among animals one of the most serious epizoötics of recent years commenced in Mauritius towards the end of 1901. The origin is attributed to the introduction of animals—probably cattle infected with the *Trypanosoma* of surra from India. So severe was the disease that between July and October of the following year over 3500 deaths in horses and cattle were recorded. The disease still remains in an enzoötic form, and frequently manifests itself among the animals of individual owners.

In areas inhabited by tsetse flies (*Glossinæ*) the disease is constantly present. These flies are fortunately confined (with one exception) to portions of Africa, but their presence precludes the possibility of stock-raising, and almost renders the use of animal transport, except at the cost of sacrifice, impossible. In Northern Rhodesia alone over seventy-five million acres are infested by this fly, and are consequently rendered useless for any purpose demanding the maintenance of animals.

Animals affected.—Each form of trypanosomiasis, due to a distinct species of *Trypanosoma*, operates upon one or more species of animal. *Trypanosoma gambiense* of human sleeping sickness has not been proved to be pathogenic to any species of domestic animal in nature, and, with the possible exception of *Trypanosoma rhodesiensi*—a form detected in man in 1909—no trypanosome normally infecting domestic animals has yet been shown to be pathogenic for man, although temporary lodgement has been demonstrated.

The Indian surra is essentially a disease of the horse and camel. Dogs when infected succumb readily; horses as a rule die in about six weeks; camels may live for three years or longer, provided they are not overworked or their vitality reduced by other means. Cattle, although they occasionally appear to succumb, may act as reservoirs for this trypanosome, and harbour infection for many years without manifesting any symptoms; no parasites will be found in their blood by microscopical examination, yet these can be proved to be present by subinoculation into more susceptible animals.

It will be seen from the subsequent discussion of species that there are some trypanosomes which are pathogenic only to horses and dogs, and which, like the trypanosome of surra, do not appear to affect cattle frequently; that there are others, such as *Trypanosoma vivax*, pathogenic for cattle, sheep, and goats which do not affect the dog, even by injection of very large doses of infected material; and there is another kind (*Trypanosoma asini*) which will affect only the horse, other domestic animals apparently being immune. *Trypanosoma congolense* of British East Africa was essentially a form infecting cattle; in equines naturally and experimentally infected there was every indication of spontaneous recovery in a large proportion.

Symptoms.—The objective symptoms manifested by a case of trypanosomiasis depend firstly upon the nature of the infecting trypanosome, and secondly, on the species of the host infected. Thus, as already noted, the infection that occurs with *Trypanosoma theileri* in oxen rarely gives rise to more than a mild pyrexial reaction of an evanescent nature: infection of the same animals with *Trypanosoma congolense* brings about a train of symptoms that may be regarded as characteristic for the disease.

In general, the picture presented by any animal infected with a trypanosome capable of causing death within about two months may be described as that of pyrexia followed by that of an anæmia. In some cases—more particularly in sleeping sickness of man—the anæmia is not particularly obvious, and the symptoms of a more chronic infection are those which indicate changes in the central nervous system.

Speaking broadly, symptoms may be classified as falling under three types, viz.:

1. Hyper-acute.
2. Acute.
3. Chronic.

Hyper-acute cases are comparatively rare, and, so far as domestic animals are concerned, are practically unknown, but there exists in East Africa a form caused by *Trypanosoma asini* which causes death of horses and mules within a few days of infection. The symptoms appear suddenly; there is marked depression, intense œdema of the genitalia and the head, and death within a few hours of symptoms being noted. Cases of this character occurred during the recent campaign in German East Africa, especially in the neighbourhood of Voi and Moschi.

Acute cases may be described as those which last from one to three weeks after infection. Here the symptoms are those of pyrexia, occasionally accompanied by œdema of the dependent parts—more particularly in the horse; changes in the eye in dogs—conjunctivitis and keratitis, with lesions of a septicæmic character on post-mortem.

The *chronic form* is that most commonly seen, and is the one which may be regarded as the most typical form of trypanosomiasis. In the early stages of the infection exacerbation of temperature and accompanying symptoms will be observed, to be succeeded later by lesions of anæmia. The working animal will manifest increasing weakness and inability to perform its normal work. The coat becomes staring and rough, and an emaciation is noticeable. In horses, filling of the limbs, and not infrequently œdema of the chest, abdomen, and, in mules, of the prepuce, are seen. The conjunctival mucous membranes are at first infected, and later become blanched, and even slightly icteric, and very commonly show well-marked petechiæ in the substance of the membrana nictitans. The appetite of all animals commonly remains, and is frequently increased. The discharge of fæces and urine continues normally, although polyuria is occasionally noticed.

In dogs an opacity of the cornea, succeeded by hæmorrhage into the anterior chamber, is a frequent occurrence with some strains of trypanosome.

Progressive weakness is manifested by a swaying movement, knuckling over, inability to rise, and later by the formation of bed-sores. Death may supervene at any time up to about a year from the time of infection, the duration varying with the species of trypanosome concerned, and the individual idiosyncrasy of the particular species of animal affected.

The normal chronic or subacute course of the disease can be hastened by the imposition of unfavourable conditions upon the animals. Military operations on the Indian Frontier and in Africa necessitating increased work and diminished rations exert a most unfavourable influence on the infected subjects, and the duration of working life is very materially reduced in consequence. Exposure to cold and wet have a similar effect, and so well is this recognized that, in countries where chronically infected animals may be bartered, it is not unusual for the customer to swim the suspects through a river and to observe the cases for a day or two afterwards. It is contended—and with much justice—that such immersion will aggravate the latent symptoms and cause the onset of manifestations of an acute systemic disturbance.

Diagnosis.—It will be observed from the foregoing that a diagnosis from a clinical picture is extremely difficult unless the action of any particular species of trypanosome can be suspected; the detection of this organism in the body fluids is the only certain method of arriving at a definite diagnosis.

Trypanosomata are not constantly present in the peripheral circulation, and examination may be made on several consecutive days before their detection is possible. Direct blood examination by means of a fresh preparation is therefore the most rapid and efficient, and if this is carried out during an exacerbation of the animal's temperature there is greater reason to hope for a positive finding.

Puncture of the lymphatic glands in order to obtain a small quantity of gland juice from the usually enlarged and œdematous glands is commonly practised for the diagnosis of human trypanosomiasis, and it may be beneficially applied to cattle, and even to horses, when difficulty is found in detecting the parasite in the blood.

Preparations made from the œdema which may occur are more usually positive than when the peripheral circulation only is examined. In rare cases it may be necessary to puncture the spinal canal in order to obtain a positive diagnosis. This is usually only practised in such forms as are manifesting changes of the nervous system.

In dourine the trypanosomes are rarely present in the peripheral blood, and a positive finding is best made on preparations obtained from the small œdematous plaques which occur in this disease, or from scrapings in the neighbourhood of the ulcerations, which are sometimes to be found on the genitalia of infected horses.

In order to ensure a positive diagnosis, as well as to provide material for experimental observation into the species of the trypanosome concerned, subinoculation into laboratory animals is very frequently necessary, and will usually be the best means of obtaining a definite diagnosis in chronic cases of infection. For example, horses and mules have been maintained at the Nairobi Laboratory for upwards of two years without manifesting any symptoms, and without showing any parasites upon direct examination of their blood. Injection of this blood to rats and dogs has demonstrated that the disease was still existent in them.

Pathology.—The symptoms of trypanosomiasis are engendered by the changes in the physiological state of the affected animal. In some forms of infection enormous numbers of trypanosomes may be seen in the circulating blood. Such an influx of organisms is commonly associated with a rise in temperature, a disturbance possibly evoked through the stimulation of the thermal centre by a *Trypanosoma* toxin. At this period the spleen and lymphatic glands are greatly enlarged, hyper-plastic, hæmorrhagic, and œdematous. Later, or at a period when the trypanosomes are numerous in the blood, these organs may return to normal size or remain only slightly swollen. The anæmia present is produced chiefly by a diminution in the number of red corpuscles, and also by a decrease in the amount of hæmoglobin present. White blood corpuscles are increased in number. In the human form of the disease, and in dourine of the later stages, histological changes in the central nervous system occur. This is essentially a proliferation of the neuroglia cells, especially around the lymph spaces of the brain or cord, and a small cell infiltration. This may be succeeded by a chronic lepto-meningitis and increase in the amount of cerebro-spinal fluid.

Treatment.—This section may be summarized by the remark that up to now no system of treatment has given more than temporary satisfaction. Various methods have been advocated, and numerous combinations of trypanocidal substances employed without lasting benefit or the elaboration of any line of treatment that can be regarded as constantly effective.

Arsenic and various arsenical preparations have, with aniline dyes, received the largest amount of attention, for it is undoubted that many of their preparations do exert a very direct effect upon the trypanosomes of the peripheral circulation. Livingstone used arsenic during his African travels; Lingard and other workers in India found that the life of an animal was undoubtedly prolonged by its use; and Holmes, also an observer in India, evolved a method of employment which has exceeded in value and permanence those obtained by any other system.[1]

Salts of mercury and antimony have also received attention, and in some forms of trypanosomiasis tartar emetic appears to have been followed by favourable results.

Trypanroth and trypanblau were introduced by Ehrlich as a result of his work on the trypanosome of mal de caderas in mice; these were followed by the introduction of methyl ("brilliant") green and other benzidine dyes, many of which, either alone or in combination with arsenical compounds, have occasionally brought about recovery from experimental trypanosomiasis in laboratory animals.

Evidence with regard to the treatment of domestic animals is less plentiful, owing partly to the difficulty of co-ordinating results obtained under field conditions, and also in Africa, to the more erratic results obtained due to the possible infection of a herd with more than one species of trypanosome, from which certain kinds of domestic animals may recover spontaneously.

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[1] The following abstract from Holmes' report on "The Treatment of Surra in Horses," in *The Journal of Tropical Veterinary Science* (vol. v. No. 1, 1910), indicates that worker's method of treatment more clearly. He says: "Arsenious acid is undoubtedly a specific for surra in horses. It must be administered in full subtoxic doses, and at intervals: and not in continuous dosage. . . . Animals cannot tolerate the requisite amount of arsenic if the dose be too frequently repeated. There is also the objection that by continuous small doses not only does the animal's body, but also the trypanosomes, acquire a certain amount of tolerance to arsenic." In order to establish the dosage Holmes experimented with a number of horses, and reports: "In our experiments it was found that ponies of about 300 lbs. could tolerate repeated doses of 1 gramme (of arsenious acid in solid form) without ill-effect; that a dose of 2 to 3 grammes was fatal for ponies of this weight; that horses weighing from 900 to 1000 lbs. could not tolerate more than 3 grammes. The arsenious acid was administered (in bolus) after feeding. Toxic dose is 2 to 3 grammes for ponies, and 4 to 5 grammes for horses. Arsenious acid administered in solution has a more toxic effect than when given in bolus. One gramme in solution is fatal for ponies weighing 250 lbs."

Details of two cases may be advantageously quoted to illustrate Holmes' method of treatment: *Pony No. C 6*—300 lbs.—3rd paroxysm 19th day of disease. 1st day, 1 gm.; 3rd day, 1 gm.; 5th day, 1 gm.; 7th day, 1 gm.; 8th day, 1 gm.; 11th day, 1·5 gms.; 13th day, 1·5 gms.; 15th day, 1·5 gms.; 17th day, 1·5 gms.; 19th day, 2 gms. No reappearance of trypanosomes. Kept under observation 10½ months.

Horse No. C 1—950 lbs.—3rd paroxysm 17th day of disease. 1st day, 1 gm.; 3rd day, 1·5 gms.; 5th day, 1·5 gms.; 7th day, 2 gms.; 9th day, 2·5 gms.; 11th day, 2·5 gms.; 13th day, 3 gms.

After this last dose of 3 gms. the horse was off his feed for three days, and treatment was suspended for ten days.

24th day, 2 gms.; 26th day, 2 gms.; 28th day, 2 gms.

No reappearance of trypanosomes. Kept under observation six months.

Later in the same report Holmes details another line of treatment. "A dose of arsenious acid in solution (Loeffler and Rueh, 1-1000) in drench was administered at a period of paroxysm. This was followed on consecutive days by a dose of atoxyl subcutaneously and a dose of arsenious acid in bolus. These three doses were repeated once or twice at intervals of four days, and once after an interval of eight days. . . . In some cases a dose of atoxyl subcutaneously was given first, which cleared the circulation of trypanosomes within 24 hours. This system of treatment has been the most successful of any tried."

As illustrating the system the following may be quoted: *Pony No. C 23*—300 lbs.—2nd paroxysm 13th day of disease. 1st day, 500 c.c. arsen. ac. sol. (1-1000); 2nd day, 50 c.c. atoxyl (4 per cent sol.); 3rd day, 1 gm. arsen. acid. Four days' interval. Doses repeated on 8th, 9th, and 10th days. Four days' interval. Doses repeated 15th, 16th, and 17th days. Eight days' interval. Doses repeated 26th, 27th, and 28th days. No return of trypanosomes. Kept under observation nine months.

With regard to camels with surra, Leese has shown that the number of trypanosomes can be kept down by the intravenous injection into the jugular of tartar emetic every three days, in doses of 25 c.c. of a 2 per cent solution. The course may thus be extended from 2 to 6 months.—G. H. W.

Immune and hyper-immune serum have been employed extensively in laboratory experiments, and also the sera of animals insusceptible to infection by particular species of parasite; organ extracts, bacterial cultures (owing to the evidence that microbial contamination rapidly destroys the vitality of trypanosomes), and killed or attenuated trypanosomes have also been utilized, without, as yet, apparently modifying the course of acute infections.

In chronic types life may be prolonged by good management and nursing.

Transmission.—At the time of Griffiths-Evans' investigations into surra in the Punjab (1880) local camel-owners ascribed the origin of the disease to bites of the *Bhara dhang*, the vernacular for *Tabanidæ*.

Rogers (1901) proved that certain species of these flies were capable of conveying infection by intermittent feeding, and Mitzmain (1914) showed that the *Tabanus striatus* of the Philippines was without doubt an active agent in the transmission of the disease there. Sergent (1905–6), working in Algeria, and Fraser and Simmons (1908) in the Federated Malay States had also demonstrated the ability of the *Tabanidæ* to act as vectors.

All these writers are agreed that the fly acts as a mechanical agent, that is to say, the proboscis, soiled by contact with trypanosome-infected blood, retains its infectivity over a short period, during which it can transmit the disease, should a succeeding bite be made upon a susceptible animal. It is probable that the proboscis can only remain infective for up to about fifteen minutes after feeding.

Mal de caderas has been shown by Sivori and Lecler (1902) capable of being spread by both *Tabanus* and *Stomoxys*, also in a mechanical manner.

In Africa, where, with one small exception, the tsetse flies alone are found, the disease *nagana*—a Zulu word, and therefore strictly only applicable to Zululand—was suspected by the natives as being caused by the bites of these flies, but it was not until 1895 that Bruce, in the course of his experiments in Zululand, was able to demonstrate the connection between the fly and the disease, which for so long had been suspected by natives living in the infested areas, and by travellers into Central Africa.

Very extensive work on the relationship between the tsetse flies and trypanosomiasis was carried out in Uganda during the course of the sleeping sickness epidemic (1903–8), when the Sleeping Sickness Commission, under the leadership of Bruce, proved conclusively that in that country the disease could be spread by the bites of a species of tsetse, *Glossina palpalis*, the distribution of which along the water-courses coincided with the existence of the disease. It became obvious as a result of their work and of the epidemiological study of human trypanosomiasis that purely mechanical transmission by means of a soiled proboscis could not entirely explain the incidence of the spread of the infection, but until 1907 all attempts to demonstrate the development of the trypanosome within the body of the tsetse failed.

In that year Kleine, working in German East Africa, in collaboration with Taute, showed that laboratory-bred flies, although demonstrated to be non-infective at birth, had acquired the property of conveying infection some three weeks after they had fed upon trypanosome-containing animals. This was speedily confirmed by Bruce and his fellow-workers in Uganda, by workers in the French and Belgian Congo, and in other parts of Africa where the tsetse fly occurred. It has since been repeatedly demonstrated that the observations of Kleine are correct for all species of trypanosomes capable of being transmitted by *Glossinæ*, and it has been further shown that the different groups of trypanosomes may each follow a different life cycle within the body of the fly. Thus, while species resembling *Trypanosoma gambiense* develop in the intestinal tract, and finally invade the salivary glands, they do not at any time develop within the proboscis. A group represented by *Trypanosoma pecaudi* develops within the intestinal tract and in the proboscis, but does not invade the salivary glands; while a third group, which includes in its numbers species resembling *Trypanosoma vivax*, all of which present slight morphological peculiarities, and are incapable of infecting dogs and small laboratory animals, develops exclusively within the proboscis.

It has been further shown that a tsetse fly once having acquired infection through a meal of blood containing a mammalian trypanosome remains infective from the time the development of the trypanosome within its body is complete up to its death. That is to say, a tsetse once infected is always infective.

Despite very numerous observations in countries where *Glossinæ* do not occur, it has not yet been demonstrated that a trypanosome can undergo any developmental cycle within the body of any other species of biting fly, but as indicated for surra and mal de caderas, the transmission of these infections through *Tabanidæ* and *Stomoxys* is mechanical.

In countries contiguous to those wherein *Glossina* occurs one is apt to disregard the importance of mechanical transmission. The culpability of the tsetse flies is so well recognized that in their absence it is considered the disease is incapable of spread. Montgomery and Kinghorn, Owen, and other writers on the disease in Northern Rhodesia, have emphasized the importance of this question, and have adduced epidemiological evidence in support of the contention that under certain conditions a trypanosomiasis of domestic stock, the origin of which was probably the bites of infected *Glossina,* may be transmitted to healthy stock in localities where tsetse fly can certainly not be demonstrated to occur. Jack, in Southern Rhodesia, Jowett in Portuguese East Africa, Aders in Zanzibar, have, among others, demonstrated that the disease can occur even where tsetse flies do not exist.

Three factors are essential for the transmission of the disease. There must exist :

1. The originally-infected host.
2. A new susceptible animal.
3. The vector or transmitting agent.

In countries where *Glossinæ* do not occur it is presumed that naturally infected domestic stock, even though they themselves are not manifesting acute symptoms of the disease, are the reservoirs from which *Tabanidæ* and *Stomoxys* acquire infection. Under the conditions of mechanical transmission it is therefore obvious that these three factors must be in close relationship one to the other ; otherwise, as has been already mentioned, the infection will die out from the proboscis of the biting fly, which becomes harmless within, say, half an hour after its infecting bite.

Surra of the camel is usually a slow disease, and as trypanosomes are commonly fairly numerous in the blood, a feed upon one of these animals is very likely to soil the proboscis of the biting fly.

Horses in the late stage of infection commonly show many trypanosomes in their blood, and owing to the lethargy and inertia engendered by the disease, biting flies will settle more readily and feed more easily upon such enfeebled animals.

The mechanical transmission of trypanosomiasis in Africa is encouraged by the methods under which the domestic animals (cattle) are kept. Owing to the presence of wild carnivora, it is customary for the animals to be put into a *kraal* or *boma* about sundown, at which period *Stomoxys* are most active. The kraal will be of varying size, but is generally not much larger than allows sufficient room for all of the animals to enter and lie down. Under these conditions mechanical transmission can be most readily effected. The *Stomoxys,* being disturbed in its meal upon a trypanosome-containing host, immediately transfers itself to an adjacent animal, upon which the meal is finished, infection being conveyed through the soiled proboscis.

No species of *Glossina* possesses the domestic habits of *Tabanus* or especially of *Stomoxys*. They are essentially flies of the wild, and usually avoid habitations.

Bruce in his Zululand work proved that certain species of game (including bush-buck, koodoo, and hyæna) were capable of harbouring the trypanosome with which he was working, although they themselves did not appear to suffer. This has been repeatedly confirmed by subsequent workers, and it is now recognized that infection is acquired through *Glossinæ* feeding upon game living in their haunts. It has never been clearly demonstrated that game does not suffer from the effects of trypanosomiasis, but it will be obvious that they must possess a very substantial resistance to it, for, speaking broadly, that type of country most favoured by *Glossina morsitans* is also favoured by the more shy and retiring species of big game, which continue to multiply despite the fly.

Considerable confusion exists in the literature with regard to the way any given species of trypanosome can be conveyed by more than one species of tsetse fly. This confusion is largely engendered by the great difficulty of identifying trypanosome species, more particularly members of morphologically similar groups, which possess varying pathogenic properties. Especially was this the case with *Trypanosoma gambiense* of man, the diagnosis of which on morphological grounds alone is almost impossible, and which, in order that final proof may be obtained, must be inoculated into man in order to demonstrate the pathogenicity to that subject. This is obviously impossible, and consequently there is considerable reason to believe that some of the work stated to have been conducted with this trypanosome was in reality carried out with one specifically distinct.

The importance of this question lay in the gradually accumulating knowledge that sleeping sickness appeared to be confined to areas where *Glossina palpalis* occurred. Adjacent to these areas, however, are in some countries enormous tracts of territory infested with *Glossina morsitans,* and, were it possible for this latter to acquire and to transmit *Trypanosoma gambiense,* the position in regard to the distribution of this disease would indeed be a serious one.

Glossina palpalis lives only in the neighbourhood of water, and under conditions of warmth, not necessary for *Glossina morsitans*. Kinghorn and Yorke demonstrated that under normal conditions of range of *Glossina morsitans* this fly did not become infected with *Trypanosoma gambiense,* but if the experiments were conducted in low-lying valleys, wherein the conditions of moisture and warmth were favourable, or if the flies were kept under artificial conditions of increased warmth—as in an incubator—they can then acquire the infection, and *Trypanosoma gambiense* will multiply and develop in them as readily as in *Glossina palpalis*.

It yet remains to be proved whether a trypanosome, which in nature occurs in countries free from *Glossinæ*, as, for example, *Trypanosoma evansi* of India, can undergo development in the tsetse fly. As bearing upon this point it may be mentioned that an East African strain of trypanosome obtained from camels coming from Abyssinia did not undergo development in *Glossina palpalis* when fed to these flies in Uganda by Duke.

It must be admitted that our knowledge of transmission is, with the exception of the known facts in regard to the development of tsetse, very unsatisfactory, for it is only reasonable to consider that should one species of trypanosome require as a definite host a particular genus of fly or other biting insect, that other species of trypanosomes should likewise require such hosts, and that the mechanical method of transmission is not the *sole* method of propagation in nature. That such a definitive host is necessary is indicated by the requirements of certain species of trypanosome found in fish which undergo their developmental cycle in various leeches, and also in the development of *Trypanosoma lewisi*, the common species of the wild rat within the rat flea. It is considered by Noeller that *Trypanosoma theileri* undergoes a developmental cycle within *Tabanus glaucopis*.

In spite, therefore, of every indication that a specific developmental cycle is undergone by every species of trypanosome within some definitive host it must not be forgotten that under the artificial conditions in which domestic stock are so frequently maintained, mechanical transmission is possible, and is, in some cases, responsible for very serious mortality.

Prophylaxis.—We have already stated that three factors are necessary for the propagation of trypanosomiasis :

(1) An infected host : a naturally infected animal, or a reservoir, such as game ; (2) a susceptible animal ; and (3) a transmitting agent.

The removal from the cycle of one of these factors will effectively check the disease.

In dealing with outbreaks of trypanosomiasis which appear to result from mechanical transmission within a herd, the early detection and segregation or slaughter of all animals found infected is strongly indicated. To this may be added measures directed against the believed transmitting agent.

Tabanidæ are mostly sylvan forms, and grazing grounds removed from their haunts could possibly be selected.

Stomoxys breeds for preference in the manure of and adjacent to cattle kraals, and, since he is most active in the evening, his worst periods can be avoided by returning the cattle to the kraals after dark, by which time he has become more quiescent. The activities of *Stomoxys* may also be controlled by burning smoky fires around the cattle, and, if these fires are composed of manure, they serve a dual purpose by destroying eggs and larvæ of the fly deposited therein.

Numerous formulæ have been suggested for dipping or spraying stock in order to ward off bites of blood-sucking flies, and where practicable there is no doubt that such measures, carried out either with arsenical or with odoriferous substances, possess some value. They are not, however, always practicable in the remote districts where *Glossinæ* occur.

It has been further stated that several species of game have been shown to harbour trypanosomes pathogenic to domestic animals, and the view is held, particularly by residents in tsetse-fly areas, that the eradication of the game would bring about a cessation of the disease, and incidentally the extinction of the tsetse fly. While there can be no doubt that measures framed against any animal known to harbour trypanosomes must, if effectively carried out, reduce the number of reservoirs, it should not be forgotten that other animals than " game " may also be capable of acting as hosts to the trypanosome. Small buck, ground hares, and other rodents are extremely numerous in many tsetse-fly areas, and it would be a physical impossibility to eradicate all such.

The disappearance of the tsetse fly from the Northern Transvaal is ascribed to the epidemic of rinderpest in 1897. Two explanations for this have been offered ; the first being that the fly was actually poisoned by the virus of rinderpest, which it imbibed in its meals upon pest-stricken game, and secondly, that owing to the diminution in the numbers of game brought about by the prevalence of rinderpest the needful food supply for the maintenance of the fly disappeared.

Experiments undertaken by the writer in British East Africa indicated that the rinderpest virus, *per se*, was not harmful to *Glossina palpalis*. The second hypothesis is in certain measure discounted by the fact that over some extensive areas in Northern Rhodesia, wherein tsetse fly is particularly prevalent, only a very limited number of game are domiciled, and it would appear from the result of observations made by Kinghorn and the writer that on occasions at least *Glossina morsitans* can feed and exist upon vegetable juices and the blood of birds.

No method has yet been devised for destroying *Glossinæ* in large numbers. Traps of various designs have been suggested, natural enemies have been sought for, as, for example, the Indian jungle fowl, which were introduced to Uganda owing to their scratching habits and their fondness for the *Glossinæ* pupæ which were unearthed, but up to the present time no satisfactory solution has been attained, nor does it, in view of extensive failures, seem likely of solution within the near future.

Zoology.—Trypanosomes belong to the class Flagellata of the order Protozoa, and as such are unicellular, simple organisms multiplying by binary fission. They are closely related to *Crithidia* and *Herpetomonas*, and, like them, possess in addition to a main nuclear element a flagellum which arises in the vicinity of a smaller nuclear structure, the centrosome, or blepharoplast. The name is derived from the Greek *τρύπανον*, a borer, and *σῶμα*, a body, a designation given on account of the method of progression observed by the earlier writers in their study of these forms in the circulating blood.

In a stained preparation the body of variable length is observed to be elongated and to be bordered on one side by an undulating membrane upon the outer border of which the chromatin-containing rim, arising from the blepharoplast and terminating in a flagellum, rests. The body plasma, in addition to the two nuclear elements mentioned, frequently shows the presence of chromogenic granules and also of vacuoles. The relative size of the body, the size and position of the blepharoplast, the development of the undulating membrane, and the length of the free portion of the flagellum are the morphological features to be chiefly considered in attempting to classify the species, and in thereby arriving at a diagnosis of the trypanosome concerned.

The smallest pathogenic trypanosome is rarely less than 10 microns in length, and 1½ in breadth at the broadest point.

Trypanosoma theileri may measure as much as 60 microns long. This is, however, a size not reached by any other species of trypanosomes found in domestic stock, among which a length of 35 microns is commonly the longest form to be encountered.

Methods of Study.—For the diagnosis of presence or absence of trypanosomes the wet film or fresh preparation possesses many advantages. Unless it is desired to continue observation for a prolonged period or to carry out this work in a very dry atmosphere, it is unnecessary to seal the edges of the cover-glass, the pressure of which alone is sufficient to cause a drop of blood to spread out between itself and the slide, so forming a thin film of temporarily uncoagulated blood in which the movement of the active trypanosome can be readily observed under the high power of the microscope, ⅙ inch or " D."

It has already been mentioned that in some conditions the examination of gland juice, obtained by puncture of a hyper-plastic lymphatic gland by means of a syringe needle, the fluid of an œdema, or the cerebro-spinal fluid obtained by lumbar punctures, may on occasions be substituted for the blood.

Centrifugalization is occasionally resorted to, the trypanosomes, after treatment of the blood in the hæmocrit attachment of the centrifuge, being present below the layer of white blood corpuscles.

Occasionally, when wet films fail to reveal the presence of trypanosomes these may be detected by the examination of films made by the thick-film method of Ross, the hæmoglobin being washed out prior to staining. The organisms of such thick film preparations are commonly greatly distorted, and do not present the typical picture shown in smears prepared by the ordinary thin-film method. In this latter staining is effected by any of the modifications founded upon the Romanowsky stain, which depends for its action upon a combination of methylene blue and eosin. Giemsa, Lieshman, Wright, and many other modifications of this stain have been introduced, the object being to contrast-stain the trypanosome whereby the blue body containing the purple nucleus and the red blepharoplast and flagellum stand out prominently. Artificial cultures of trypanosomes have been effected, but is not a common practice of any laboratory as a means of diagnosis or maintenance of the strain.

For animal inoculation experiments, blood is extracted from the host either by snicking the ear or the tail, as in the case of rats ; the blood that issues being collected in a capillary pipette, from which it is immediately transferred to a citrate solution to prevent coagulation ; or if a larger quantity is required it is obtained by puncture of the jugular vein, and collected in a syringe or other vessel. The material so obtained can be injected into the new host by subcutaneous, intravenous, or intraperitoneal methods ; and, should the trypanosome be pathogenic to the injected subject, infection will commonly be declared after an *incubative period of five to fourteen days*, a period dependent largely upon the number of trypanosomes injected, and the method of injection.

Diagnosis of Species.—Two principal factors are involved in the classification of trypanosomes :

1. Morphology, which considers shape, size, and development of the blepharoplast, undulating membrane, and free flagellum.
2. Pathogenicity, which considers the sus-

ceptibility or insusceptibility of various species of animals for the particular trypanosome.

Among the trypanosomes occurring in the tsetse-fly areas of Africa rudimentary classification may also be effected by observing the fashion in which multiplication takes place within the body of the tsetse fly. As already mentioned, some appear to develop within the intestinal tract, and subsequently invade the salivary glands. Others develop in the proboscis and the intestinal tract, and *not* in the salivary glands, and others again multiply only in the proboscis.

This last system of classification, however, is of little value to a man removed from laboratory facilities; indeed it must be stated that comparatively little work of a rigorous character can be attempted by any one who is removed from the facilities of isolation for his experimental animals and opportunities of observing these in large numbers.

The animals most commonly associated with experiments in trypanosomiasis are white mice, white rats, guinea-pigs, and dogs. Rabbits, owing to their relative insusceptibility, are less commonly used. Sheep, goats, and the larger domestic animals should be employed wherever possible. Owing to the difficulty of obtaining the small laboratory animals in some countries, small mammals captured in the wild state have been utilized.

To a worker entirely removed from facilities for animal experimentation a classification on grounds of morphology alone is more desirable. By this means a very good approximate idea of the trypanosome concerned can be formulated, but wherever possible this *must* be substantiated by animal experiment, which alone can answer the question of pathogenicity.

By a combination of morphological and pathogenic study it is possible to place any detected trypanosome within a group, if not to assign to it a definite specific name. We do not know exactly what degree of variation from an accepted normal should be permitted in any form without necessitating the creation of a new species, and it is doubtless for this reason that the past fifteen years has witnessed the creation of a great number of so-called "species," the majority of which have been detected in Africa.

In earlier work upon this disease, observers, having detected what they regarded as a new trypanosome, despatched an infected animal to a European country removed from the natural disease, where the organism could be studied under rigorous laboratory conditions. By so doing it has been possible to furnish very accurate information with regard to such forms as *Trypanosoma evansi*, *Trypanosoma gambiense*, *Trypanosoma equinum*, *Trypanosoma equiperdum*, and a few others. *Trypanosoma brucei* was also studied in Europe, where the strain, originating in Zululand in 1896, is still maintained.

Workers in Africa have been constantly faced with the difficulty of obtaining experimental animals; with the danger of these becoming naturally or accidentally infected with more than one strain of parasite; and with the fact that in a tsetse-infected area, where several species of trypanosomes unquestionably occur, tsetse flies infected with several species of trypanosomes may at successive bites transmit one or more of those species to different animals. In the course of an outbreak of the disease amongst domestic stock, therefore, the detection of any one species of trypanosome should not be accepted as being the only agent involved in the epidemic. Considerable confusion, too, may arise from the fact that an experimental animal inoculated with blood showing a particular form under the microscope may develop a trypanosome possessing entirely different morphology. One possible example of this may make the case clear. *Trypanosoma nanum*, a particularly small form, may be observed in the blood of cattle, and, as the result of an inoculation into a rat or guinea-pig, a trypanosome morphologically indistinguishable from *Trypanosoma evansi* may appear in the blood of that rat or guinea-pig. This is due to the fact that *Trypanosoma nanum* is incapable of infecting those small animals, and that the ox *may*, in addition, have been infected with *Trypanosoma evansi*, which very rarely shows itself on direct microscopical examination in that species. There can be little doubt that errors of this nature have occurred in some of the earlier work upon trypanosomiasis in Africa, and they are errors very difficult to guard against unless the worker is possessed of extensive facilities for this class of investigation.

By accepting the morphological appearance as a means of identification it is possible to identify *Trypanosoma theileri* on account of its great size, and *Trypanosoma equinum* owing to its peculiarity in possessing a most rudimentary blepharoplast, and, with certain reservations, *Trypanosoma rhodesiense*, which, in some forms, shows an unusual distribution of the relationship between the nucleus and the blepharoplast within the body.

On morphological grounds groups of trypanosomes represented by *Trypanosoma evansi*, in which the flagellum is always long and free, and *Trypanosoma congolense*, in which it is invariably absent, may be constituted. The group represented by *Trypanosoma vivax* may also be recognized by the blunt posterior extremity of the body and the very poorly developed undulating membrane.

By accepting pathogenicity as a standard it is possible to eliminate *Trypanosoma gambiense* and *Trypanosoma rhodesiense*, which, of all known species of true trypanosomes, alone infect man, and *Trypanosoma equiperdum* of dourine, which, under conditions of nature, is transmitted almost exclusively by coitus, and invokes a train of symptoms quite distinct from that usually caused by other species, and it is possible to place a given organism within a group. For example, *Trypanosoma lewisi* is pathogenic only for rats; *Trypanosoma theileri* only for cattle; *Trypanosoma vivax* and its relatives will not infect dogs or the small laboratory animals; *Trypanosoma simiæ* is pathogenic for sheep, goats, pigs, and monkey, but is not inoculable to oxen, dogs, and small laboratory animals.

In addition to the absolute pathogenicity of a given species the relative pathogenicity may also be considered as an aid to specific identification of a form.

Other aids for classification exist, *e.g.* agglutinating properties of different sera; method and facility of culture under artificial conditions; and, most important, the method of "cross inoculation," employed by Laveran and his co-workers. This method is based upon immunity or insusceptibility to a particular species or strain of trypanosome, as demonstrated by spontaneous recovery and subsequent resistance to re-inoculations with the same strain. The infection of such an animal by inoculation with another strain of trypanosome is held by Laveran to justify the recognition of the latter strain as a distinct species. In this way *Trypanosoma soudanense*, *Trypanosoma berberum*, *Trypanosoma morocanum*, and others have come into being.

The following table indicates a method whereby a trypanosome may be placed in its group class. It must, however, be clearly understood that there do exist named species, the position of which, owing to incomplete data regarding them, it is difficult to define. It is also to be noted that the writer's interpretation of some of the different species referred to is not that accepted by all workers.

TABLE OF CLASSIFICATION

A. Trypanosomes in which the flagellum is always free:
  (*a*) and which produce a rapidly fatal infection in the usual laboratory animals; Group type *Trypanosoma evansi*;
  (*b*) and which are non-pathogenic to the usual laboratory animals; Group type *Trypanosoma vivax*.

B. Trypanosomes in which the flagellum is never free:
  (*a*) and which usually produce a fatal infection in laboratory animals; Group type *Trypanosoma congolense*;
  (*b*) and which are non-pathogenic to the usual laboratory animals; *Trypanosoma nanum*.

C. Trypanosomes in which the flagellum is sometimes free, and sometimes absent (di- or polymorphism); Group types *Trypanosoma dimorphon* (*sensu* Dutton and Todd), and *Trypanosoma pecaudi*.

Specific Study of Species

1. GROUP *Trypanosoma evansi*

With the exception of *Trypanosoma equinum*, in which the blepharoplast is extremely small or invisible, the species of this group present a marked morphological resemblance.

On pathogenic grounds *Trypanosoma equiperdum* may be excluded in so far that it is transmitted naturally by coitus: and further that some laboratory animals are more resistant to it than to other members of the sub-group.

Trypanosoma hippicum is stated to possess a free flagellum, shorter than is common with *Trypanosoma evansi*. This species has only been found near the Panama Canal.

Trypanosoma soudanense, *Trypanosoma annamense*, and *Trypanosoma morocanum* are distinguishable from *Trypanosoma evansi* only by immunity experiments.

Trypanosoma evansi and *Trypanosoma brucei* are said to be discernible by careful comparison, but, in general, their morphology and animal reactions are peculiarly similar. In the past ten years some authors, in particular Bruce and his colleagues in Uganda and Nyasaland, have described as *Trypanosoma brucei* forms of trypanosomes distinctly dimorphic, and not at all answering to the original description of this species by Plimmer and Bradford, a description based on the strain originating in Zululand, which is still maintained in some European laboratories.

To be regarded as *Trypanosoma brucei*, or to fall into the *evansi* sub-group, a trypanosome must be monomorphic, and must carry a free flagellum. Immediately after division it is possible for an occasional non-flagellated form to occur, but such are markedly rare, and a biometrical count will show their insignificance.

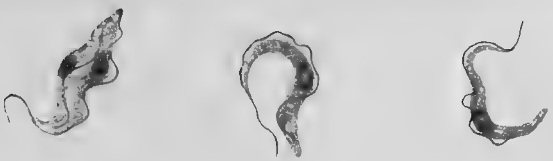
Morphology of the Trypanosomes of this Group. *In Fresh Preparation.*—Translatory movement is almost invariably restricted to passage within the area of the field of the microscope; it does not enter and cross as in the case with species of the *vivax* group. Undulating activity is marked, and the long, free flagellum greatly disturbs the corpuscles.

In quiescent forms the details of undulating membrane and flagellum can be clearly made out.

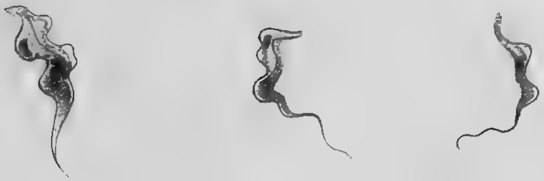
In Stained Preparation.—The trypanosome measures from 17 to 36 microns in length, including the free flagellum. The width varies from 1·5 to 2·25 microns.

The body is elongate, the posterior extremity pointed or bluntly conical, and the cytoplasm, especially in animals showing a heavy infection, frequently contains granules.

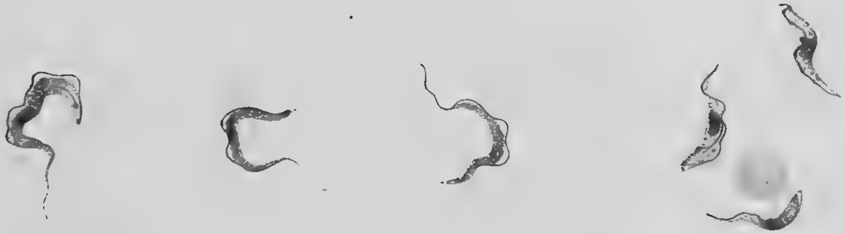
The blepharoplast is small and distinct, and sometimes removed two or three microns from the extremity.



Trypanosoma evansi (Surra).

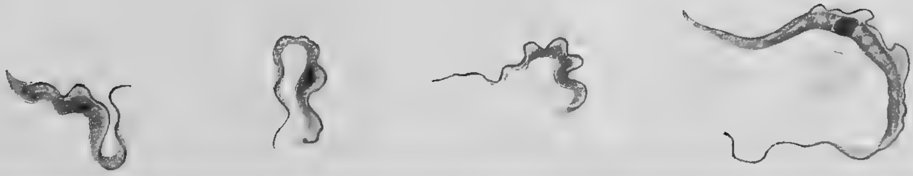


T. equinum (Mal de Caderas).



T. brucei (Nagana).

T. congolense.



T. equiperdum (Dourine).

T. theileri.

FIG. 33.—Trypanosomes. Zeiss oil immersion $\frac{1}{12}$ in., eyepiece 4. (After Martin Mayer.)

The nucleus is round or oval, and situated about the middle of the body.

The undulating membrane is well developed, and shows a number of definite plications of varying sizes.

A free flagellum exists on over 95 per cent of the trypanosomes encountered. A number, varying from 1 per cent to 4 per cent, are met with in nearly all slides in which no such structure can be determined; these are probably forms resulting from a recent divisional multiplication. The free portion of the flagellum varies from 2 to 10 microns in length; the average is about 5 microns.

Pathogenicity of the Trypanosomes in this Group.—Excluding *Trypanosoma equiperdum*, the causal agent of dourine, the other forms are pathogenic to equines, dogs, guinea-pigs, and rats. The organisms are intermittently present, sometimes in large numbers, but in cattle, sheep, and goats this group of trypanosomes has less effect. The inoculation of ruminants with large doses of trypanosome containing blood may be followed in five to seven days' time by a sharp exacerbation of temperature and the appearance of a few organisms in the blood. From that time parasites disappear, and are usually never again seen under direct microscopical examination. A small percentage of infected animals will manifest symptoms of the chronic form of trypanosomiasis, and may succumb to this, more especially in animals subjected to overwork or other debilitating influences, such as a heavy invasion of the intestinal parasites.

The effect of *Trypanosoma evansi* upon cattle was studied by Nocard and Vallée, who in Paris injected a number of cattle, only one of which succumbed. In India cattle and buffaloes are regarded as reservoirs of the disease, but when the infection was introduced into Mauritius a large number of cattle succumbed.

Trypanosoma brucei in Europe behaved in a very similar manner to *Trypanosoma evansi* when inoculated into cattle; thus Nocard injected several Breton cows, none of which succumbed. Lignières obtained similar results in Argentine cattle. This feature is worthy of comment in view of the fact that Bruce describes cattle as being susceptible to *nagana* of Zululand, and it may be taken as evidence that this authority was working with more than one strain of trypanosome during his investigations (1894–96), and that some of his experimental work was carried out with a trypanosome specifically distinct from that sent to Europe, and later called *Trypanosoma brucei*.

Although ruminants rarely show trypanosomes of this group in the peripheral circulation, their blood may be infective for many months in spite of apparent good health. Thus, in the course of an experiment carried out in East Africa, an ox, which was inoculated in May 1915 with the blood of a horse showing numerous trypanosomes of this group in its blood, never showed objective signs of infection, but by subinoculation its blood was proved to be infective on the 29th, the 174th, and the 317th days after inoculation, but non-infective on the 442nd day, and the animal was still alive in June 1917, the 762nd day, and had gained 80 kg. in body-weight during the period of observation.

2. GROUP *Trypanosoma vivax*

The species falling into this group are *Trypanosoma vivax*, *Trypanosoma cazalboui*, *Trypanosoma uniforme*, *Trypanosoma capræ* (*sensu* Bruce and others), and *Trypanosoma bovis*, Kleine.

It is a point for academic discussion whether *Trypanosoma vivax*, Ziemann, is or is not a true species. In its original description certain observations were made, both as regards the morphology and the pathogenicity, which are not now admitted even by the supporters of the species. The same may be said of the early observations on the trypanosome first encountered by Cazalbou in the disease *La Soumaya*, from a case of which the trypanosome subsequently reached Paris and enabled Laveran to publish precise and uncontestable data and to create the valid species *Trypanosoma cazalboui*.

There is no doubt that *Trypanosoma cazalboui* is not identical with the form described by Ziemann, since specimens made by that writer have been available for subsequent examination and measurement, and have been found to differ materially in size. Further, workers in the French Guinea, in Erythrea, Uganda, Northern Rhodesia, and German East Africa have also met with trypanosomes of the distinctive morphological type which were too large to be regarded as *Trypanosoma cazalboui*.

Trypanosoma uniforme is a species clearly associated with the group *vivax*, and differs from *Trypanosoma vivax* and *Trypanosoma cazalboui* in being smaller than either.

Trypanosoma capræ was described by Kleine as affecting sheep and goats only. In subsequent papers this author, and also Fischat, points out that *Trypanosoma capræ* is dimorphic, occurring as a free flagellated form which may measure 31 microns, and also as a non-flagellated form of only 18 to 20 microns.

Bruce and his colleagues in Nyasaland describe as *Trypanosoma capræ* a trypanosome found in that country, in which all forms showed a free flagellum. They also note that oxen were receptive to their trypanosome, whereas Kleine had stated that oxen were insusceptible. From this it appears possible that the Nyasaland Commission were not dealing with Kleine's *Trypanosoma capræ*, but with a different species.

Morphology.—The measurements of the trypanosomes of this group as given by respective writers are as follows :

Trypanosoma vivax—

Togoland, 18 to 26, even to 30 microns (Ziemann's measurements).
Togoland, 23 to 31 microns (measurements by Bruce and others).
French Guinea, 26 to 28 microns in horses (G. Martin).
French Guinea, 22 to 28 microns in cattle (G. Martin).
Uganda, 20 to 27 microns (Bruce and others, 1903 strain).
Uganda, 16 to 29 microns (Bruce and others, 1909 strain).
Northern Rhodesia, 20 to 26 microns (Montgomery and Kinghorn).
Northern Rhodesia, 19 to 29 microns (Kinghorn and Yorke).
Belgian Congo, up to 31, average 24·4 microns (Rodhain and others).

Trypanosoma cazalboui—21 microns (Laveran).
Trypanosoma uniforme—12 to 19 microns, average 16 (Bruce and others).
Trypanosoma capræ—18 to 32 microns (*sensu* Bruce and others).

The trypanosomes of this group which have been encountered at the Nairobi Laboratory in animals coming from different parts of East Africa, Uganda, and German East Africa fall on biometrical grounds into three distinct classes, which, perhaps curiously, coincide with origin from three distinct foci of infection :

(*a*) Origin Uganda, 15 to 22 microns, average 18 microns.
Diagnosis=possibly *Trypanosoma uniforme.*
(*b*) Origin British East Africa, 16 to 28 microns, average 23 microns.
Diagnosis=possibly *Trypanosoma cazalboui.*
(*c*) Origin German East Africa, 21 to 35 microns, average 29 microns.
Diagnosis=possibly *Trypanosoma vivax* or *Trypanosoma capræ.*

All species of this group present similar structural characters in addition to the free flagellum. The body cytoplasm is more clear and alveolar than in other groups ; the blepharoplast is commonly large and distinctly globular ; the undulating membrane is narrow and poorly developed ; the posterior extremity is commonly bluntly rounded.

In fresh preparation a peculiarity of each member of the group is the rapidity with which it crosses the field of the microscope. The intensity of this movement varies somewhat, but in some forms is so great that only the "wash" of the displacement through the corpuscles can be detected, the trypanosome itself being only visible when movement has slowed down after the preparation has been made for some time. Vibratory and undulating movements are restricted.

3. Group *Trypanosoma congolense*

In the species of this group no free portion of the flagellum occurs ; most of the members are pathogenic to ruminants ; their effect upon equidæ is less constant, and in rabbits, guinea-pigs, and rats the pathogenicity is erratic.

The forms included here are :

Trypanosoma congolense, *Trypanosoma dimorphon* (*sensu* Laveran and Mesnil), *Trypanosoma pecorum*, *Trypanosoma montgomeryi*, and *Trypanosoma simiæ*. Edington's trypanosome of Zanzibar, and Theiler's organism obtained from the Zambesi, should also be included in this group.

Trypanosoma congolense and *Trypanosoma pecorum* are not distinguishable on morphological grounds, and the animal reactions are very similar ; they are separable, according to Laveran, by cross immunity tests. *Trypanosoma dimorphon* of Laveran and Mesnil may show forms longer than the other two species.

Trypanosoma simiæ is pathogenic only to monkeys and pigs : *Trypanosoma montgomeryi*, though of similar length to *Trypanosoma congolense*, is exceptionally broad.

Fresh Preparation.—There is very little translatory movement : the trypanosome remains localized, but manifests rotatory and vibratory movements. It may often remain quite stationary, and is therefore difficult of detection.

Stained Preparation.—The body is short, measuring from 10 to 21 microns in length, and is usually deeply and homogeneously stained. The posterior extremity is rounded or bluntly conical : the anterior extremity is slightly attenuated along the rim of the undulating membrane. There is no free flagellum. The blepharoplast is rounded, and placed close to the extremity. The nucleus is round or oval, and situated about the middle of the body. An achromatic area is not infrequently seen near the blepharoplast. The undulating membrane is rudimentary, and thrown into one or two small folds only. It rarely stands out from the body.

Trypanosoma nanum.—*Trypanosoma nanum* is a species morphologically very similar to *Trypanosoma congolense* : it is non-pathogenic to dogs, rabbits, and small laboratory animals.

First observed in the Sudan, this species has subsequently been described from Uganda and Northern Rhodesia. Owing to the resemblance to *Trypanosoma congolense*, direct microscopical examination, when experimental inoculations could not be carried out, might cause a diagnosis of this latter to be given.

Trypanosoma nanum is pathogenic to ruminants : its effect upon equidæ is not known.

4. Group Types *Trypanosoma dimorphon* (*sensu* Dutton and Todd) and *Trypanosoma pecaudi*

The species *Trypanosoma dimorphon* was created for a trypanosome studied by Dutton and Todd in the Gambia. Work in the Gambia and observations in Europe did not correspond, and there now seems to be no doubt that the investigations of Dutton and Todd were carried out upon a strain of trypanosome distinct from that with which Laveran and Mesnil worked.

Trypanosoma dimorphon, *sensu* Dutton and Todd, is a polymorphic organism showing itself in three forms :

(*a*) A "tadpole" form ; very short and without a free flagellum.

(*b*) A "stump" form, with a fairly well-developed undulating membrane ; longer and broader than the "tadpole," and also devoid of a free flagellum.

(*c*) A "long" form with a free flagellum.

Trypanosoma dimorphon, *sensu* Laveran and Mesnil, does not show any free flagellated forms ; their "long" type being structurally similar to the "tadpole" form, but of greater length—up to 22 microns, their "tadpole" form of 10 to 15 microns being similar to that of Dutton and Todd.

The confusion which exists owing to two obviously different trypanosomes possessing the same specific names is considerable ; by right of priority the name should be confined to the form answering the description of Dutton and Todd, but the supporters of the opposing view consider that these workers were misled by a mixed infection of two distinct species of trypanosomes. If this be correct the name should be expunged. The question of nomenclature is an academic one, but in the interests of trypanosome workers in Africa it is most desirable that the position of classification arising from it should be explained.

Trypanosoma pecaudi is a dimorphic species, showing itself in two forms :

(*a*) "Stumpy," of 14 to 20 microns in length, with a well-developed undulating membrane, and without a free flagellum.

(*b*) "Long," measuring 25 to 35 microns long, and possessing a well-marked free flagellum.

Originally discovered in Senegal, it is probable that this species, or very closely related forms, has a wide distribution through Africa, where it has probably been confused with and described under the name *Trypanosoma brucei*.

Pathogenicity.—*Trypanosoma dimorphon* of Dutton and Todd is pathogenic to all domestic and laboratory animals, invoking the acute and subacute disease in most. In the horse and donkey it is more chronic, and natural recovery may be observed.

Trypanosoma dimorphon of Laveran and Mesnil resembles *Trypanosoma congolense* in its action ; equidæ being more resistant than other species.

Trypanosoma pecaudi.—Ruminants rarely show this form in their blood ; they are susceptible to inoculation, and trypanosomes may be seen after five to ten days' incubation. The duration of infection in these animals, as demonstrated by subinoculations into susceptible subjects, is long. Symptoms are comparatively rarely shown, and spontaneous recovery is not infrequent. In equines, dogs, and small animals the infection is acute and subacute ; trypanosomes being numerous in the blood, and the temperature is paroxysmal.

In fresh preparation, translatory, undulatory, and rotatory movements are active, and distinctly recognizable. Rarely a form crosses the field like a member of the *vivax* group. In the free flagellated forms active lashing with the flagellum is a noticeable feature.

R. E. M.

SURRA

Surra is the name now in general use to designate the diseased condition produced by the protozoal organism *Trypanosoma evansi*.

The word "surra" is an adaptation of the Hindi word meaning "rotten," which is one of the numerous names applied to the disease as it occurs in India.

Historical and Distribution.—Prior to 1880, when the cause of the disease was discovered, little or nothing had been written regarding it. The credit of discovering the trypanosome and of giving a very accurate account of many of the characters of the disease belongs to Evans. Although attention had been drawn to surra as mainly, if not exclusively, a disease of horses, Evans in his investigations at Dera Ismail Khan in the Punjab discovered that mules and camels were also attacked under natural conditions. With the materials and apparatus at his command Evans was unable to give an exact description of the morphology of the organism, but specimens were submitted to Crookshank, who in 1886 published an account of the parasite and classified it. Evans' discovery was soon confirmed. In 1885 Steel discovered an identical parasite in the blood of mules in Burmah. In the meantime numerous outbreaks of the disease were reported in India, mainly in the Punjab. In 1899 Lingard reported that the disease had been long known in many parts of India—Assam, North-West Provinces, Bombay, Burmah, and elsewhere. In the same year the occurrence of surra in the Dutch East Indies was established, and in 1901 a disease very closely resembling it was discovered in Annam.

Soon after the occupation of the Philippine Islands by America attention was drawn to the existence of surra there. In the Emergency Report published by the Bureau of Animal Industry in 1901 it is stated that the point was still undecided as to whether the disease had been recently imported or not. The view of veterinary surgeons in the islands was that it was not a recent importation and that it had previously been recognized under the name Callentura. The investigators sent by the United States held, on the other hand, that the disease was of recent introduction, having been imported with Chinese cattle, which in turn had acquired the infection from cattle introduced into China from India. It would appear that the question must remain undecided.

In 1901 the disease made its appearance in Mauritius. In this case there can be no doubt that it was imported from India with some cattle. Prior to 1901 the farmers of Mauritius had been in the habit of obtaining cattle from Madagascar, but apparently during the South African War the activity of the military authorities in purchasing cattle virtually closed the Madagascar markets to general export, with the result that the farmers of Mauritius were compelled to obtain their animals elsewhere. A number of deaths occurred during the voyage among animals brought from India. Post-mortem examination failed to reveal any cause of death, and after a little delay the surviving cattle were allowed to land. Deaths continued to occur among the imported animals, and within a short time deaths began to occur among the horses, cattle, mules, and donkeys belonging to the island. It was not until the disease had been in existence there for nearly six months, by which time a very large proportion of the animals had become affected and had died, that the real nature of the disease was established by Lesur. The disease continued to take heavy toll of the live stock until, by the end of January 1903, there was scarcely a horse or a mule left alive.

In 1906 surra was conveyed to the United States in some zebus from India. The disease was diagnosed and prompt slaughter of the animals suppressed it.

A year later surra was imported into Australia in a batch of camels from Karachi. Evidence was obtained in this instance of the long periods that may elapse without trypanosomes being discoverable in the blood of infected camels.

Within recent years there have been discovered diseases caused by trypanosomes in Africa which there are good reasons for believing are identical with surra. Cazalbou has described a disease which occurs along the whole of the northern part of Africa in horses, camels, and dromedaries, and is locally known as El Debab.

In an outbreak of trypanosomiasis in horses in Egypt it was thought that the source of infection was camels suffering from El Debab.

Mbori is another local name applied to a trypanosomiasis of dromedaries which occurs as far south as Senegal. There appears to be no doubt that this condition is also identical with surra.

The discovery of a trypanosome in animals infected with El Debab was made by Chauvrat in 1892, and the existence of this organism was subsequently confirmed by numerous other observers.

Cazalbou made the discovery of trypanosomes in dromedaries suffering from Mbori in 1903, and subsequently published a number of papers on the same subject.

It would appear that the trypanosomiasis of dromedaries in Somaliland described by Martoglio is also identical with surra.

Causal Organism.—The parasite that is responsible for surra is the *Trypanosoma evansi*. The trypanosome is somewhat variable in length but it is never dimorphic. The length ranges from 18 microns to 35 microns and its width from 1½ to 2½ microns. The average size of the parasite as it occurs in the blood of horses is about 26 to 27 microns. All the morphological characters common to trypanosomes in general are present in *Trypanosoma evansi*. In fresh preparations of blood the trypanosome is extremely active, and can be watched travelling with considerable rapidity about the field under the microscope. As in the case of other trypanosomes, multiplication takes place in the animal host by longitudinal division. There is no evidence of sexual distinction.

Outside the animal body the trypanosome of surra dies somewhat rapidly, the vitality being retained for shorter periods the higher the temperature at which the blood is kept. It has been shown that trypanosomes exposed to the temperature of liquid air for a quarter of an hour are still capable of causing infection when inoculated into susceptible animals.

Little or no success has attended efforts to obtain cultures of the parasite on artificial culture media. In one or two instances it has been found possible to obtain a small degree of multiplication in original tubes, but attempts at sub-cultivation have almost invariably failed. Parasites obtained in artificial cultures have always been found to be devoid of virulence, and the inoculation of such cultures has failed to produce any degree of immunity.

Animals susceptible.—Under natural conditions equines, camels, dogs, cattle, buffaloes, and elephants may be infected with surra. Under experimental conditions all the above species can be infected, as well as the ordinary small laboratory animals. Birds are refractory to inoculation.

Symptoms.—Horses, donkeys, and mules are very susceptible to surra, and outbreaks of the disease are not infrequent.

In horses experimentally infected there is a period of incubation which ranges from about four days to a fortnight, but usually from five to seven days. The first indication of infection is a rise of temperature, which is associated with the presence of trypanosomes in the blood in small numbers. These rapidly increase as the temperature rises and after two or three days decrease again as the temperature falls. They may remain undiscoverable in the blood by microscopic examination for a number of days, but reappear when the temperature rises again. The fever is therefore intermittent, the exacerbations of temperature corresponding more or less closely with the appearance of trypanosomes in the blood. Within a day or two of the appearance of trypanosomes in the blood petechiæ are observed on the mucous membranes, particularly of the eye. This is usually associated with lachrymation. In spite of maintenance of appetite the animal becomes weaker, and œdema of dependent parts is noticed. As the disease progresses the mucous membranes become pale and not infrequently acquire a yellowish tint. The disease, if left to run its course, is invariably fatal.

In experimentally infected horses surra may cause death in from two to six weeks after inoculation.

In natural cases death may be delayed until the third month after the first appearance of trypanosomes, but as a rule death occurs before the end of the second month.

Surra in camels is also known as " Tibarsa," which means " three years disease," or " purāna," meaning " old " or " chronic." Camels of all ages are susceptible to natural infection.

The disease is extremely insidious in its onset, and a camel may be infected for a considerable time before it is noticed to be ill. This is particularly the case if the feeding is good and the work not hard. It needs very intimate knowledge of the camel in health and disease for the recognition of the earliest symptoms of the disease. If daily records of temperature are taken short attacks of fever are detected. Close observation during these periods will show a certain amount of listlessness, the coat is rather harsh, the eye dull, and in some cases there may be some lachrymation. The urine decreases in amount and has a high colour. The mucous membranes may become pale and show petechiæ, but this is not invariably the case. The attacks of fever are longer and more frequent in the early stages of infection. They may occur once a week at first, but they gradually decrease in duration and severity as the intervals between them become longer.

In severe cases the symptoms make their appearance more rapidly, wasting and weakness are more pronounced, and finally paralysis sets in. Œdema of dependent parts is not common, but it does occur in the later stages of severe cases.

In severe cases the disease terminates fatally within a few months, but in cases of milder nature it may run a very chronic course, the affected animals remaining alive and in fair condition for years even. The mortality is very heavy. It is believed by native camel-owners that if the chronic cases survive for three years some of them will make a complete recovery.

Surra in Dogs.—Not infrequently outbreaks of surra have occurred among canines. Particular attention has been drawn to them owing to their having involved packs of hounds imported from England. One such outbreak occurred amongst hounds in Bombay, and at least two outbreaks have caused very serious losses among the hounds of the Lahore Hunt.

Dogs are easily infected experimentally by inoculation, and the symptoms in natural and experimental infections are practically identical. There is loss of appetite associated with febrile attacks ; œdema of the head and throat, swelling of the joints, and opacity of the cornea are frequently seen.

Imported dogs die as a rule within a month after experimental infection, but the native pariah dog very frequently lives for months.

Surra in Cattle.—Cattle possess a far higher degree of resistance to surra than equines. They may appear to be perfectly healthy in spite of the presence of trypanosomes in their blood. As a rule these are present in small numbers only, and their presence can often be detected only by the experimental inoculation of other animals with their blood.

Indian hill cattle not infrequently harbour the parasite in their blood in small numbers without evincing any evidence of disease, but when inoculated experimentally with blood of hill cattle or buffaloes containing the parasite they may show evidence of infection in febrile attacks, the trypanosomes being present in their blood in large numbers. On the other hand, such inoculations may lead to no evident disturbance of health although trypanosomes may be numerously present in the blood.

The susceptibility of **buffaloes** appears to be very variable. In Northern India, where surra is more common, the susceptibility of the buffalo is low, symptoms seldom presenting themselves. Many buffaloes harbour the parasite in their blood in small numbers without presenting any clinical evidence of infection. In southern

India (Madras) and in the East Indies fatal cases of surra in buffaloes do occur.

Surra in Elephants.—In elephants surra follows a chronic course, and in general the symptoms closely resemble those of chronic surra in other species.

Parasites are often present in the blood in large numbers. The disease usually terminates fatally, but cases of real recovery appear to occur.

Methods of Infection. Experimental.—Inoculation with a trace of blood containing living trypanosomes into a susceptible animal, whether subcutaneously, intravenously, or intraperitoneally, will produce the disease with almost mathematical certainty. The administration of infective material by the mouth, provided there are no lesions on any part of the buccal mucous membrane, always fails.

Natural Methods of Infection.—The fact that fresh cases of surra in horses do not occur throughout the year indicates that it is not a contagious disease in the ordinary sense of the term, and that some outside agency plays a part in its propagation. From the earliest times flies have been suspected, and all the evidence that has accumulated since proper investigations were first carried out goes to prove that this is actually the case.

In other words, surra is a disease showing a seasonal distribution; its occurrence being closely connected with the *rainy period*, which is also what may be called the *fly season*. Roughly speaking, the monsoon breaks in India about the middle of June and the rains continue up to the middle or end of September. Prior to onset of the rains biting flies of many species make their appearance, and shortly after this cases of surra begin to be observed. Cases continue to occur until October, after which fresh outbreaks are not as a rule encountered.

Experiment and experience show that more than one species of biting fly is capable of carrying surra infection from animal to animal. It has been proved that flies belonging to the following genera are able to effect this: *Tabanus*, *Hæmatopota*, *Stomoxys*, and others. Of these the *Tabanidæ* appear to be by far the most important. In at least one instance the evidence appeared to indicate that a fly belonging to another genus, viz. *Lyperosia*, is capable of acting as the transmitting agent of surra from camel to camel.

The Maintenance of the Virus.—In view of the fact that cases of surra in the horse are invariably fatal within a month or two, and that outbreaks of the disease occur in places where camels, which, as already stated, may harbour the parasite for two or three years, are at the most encountered only occasionally, there must be some source from which the flies are able to get the virus to set up an outbreak of the disease among horses. This "reservoir" is to be found in cattle and buffaloes. The power possessed by these species of remaining in apparently perfect health in spite of infection with surra has already been referred to. Once a horse has been infected by a fly which has obtained its virus from a bovine or a buffalo the disease may spread with rapidity from horse to horse, because trypanosomes become very numerous in the blood of affected horses.

Contrary to what is known to be the case in the transmission of some of the other forms of trypanosomiasis, there is no evidence to show that the trypanosome of surra undergoes any cycle of development in the transmitting flies. There is, indeed, abundant evidence to show that they do not. In other words, the fly acts merely as a mechanical agent in carrying the virus from an infected to a healthy animal.

Natural Infection in the Dog.—There appears to be good reason for believing that surra in canines is not always due to the bite of an infective fly.

Outbreaks of surra among packs of hounds are possibly due to the devouring of infected jackal, the actual infection taking place through small wounds or abrasions of the mouth. On the other hand, cases are on record in which individual dogs became infected through the bites of flies.

From what has been said regarding the transmission of surra through the medium of biting flies, it will be gathered that the disease has not only seasonal but also a regional distribution. For the majority of the flies concerned a certain degree of moisture and shade are necessary. Leese's investigations have shown, however, that it may occur in arid areas in the Punjab. In this instance the fly responsible appeared to be *Lyperosia*.

Post-mortem Appearances.—There are no distinct pathognomonic lesions discoverable in animals dead of surra. The only lesions are those generally associated with anæmic conditions. A pronounced feature of the carcase is its emaciated appearance, and in some cases dropsical swellings in dependent parts. In the abdominal and thoracic cavities there may be a variable amount of dropsical transudate, and the whole of the organs have a blanched or somewhat yellowish tint which is due to coloration with bile. The lungs are usually normal, but in some cases there is evidence of slight bronchitis. The fat in the furrows round the heart is generally replaced by a gelatinous material and the heart muscle itself is flabby. The pleura appears to be normal. The abdominal organs are anæmic and dropsical but are otherwise normal in appearance. The mucous membranes throughout the body may show petechiæ, but these are not commonly found in the digestive tract. Authorities differ regarding the lymphatic glands of the body. According to some they are enlarged and œdematous, and according to others their appearance is normal. A gelatinous œdema is present in the subcutaneous and intermuscular connective tissue.

Diagnosis. **In Horses.**—An absolute diagnosis cannot be based upon clinical observation only, but a very shrewd suspicion may be raised when the locality, season, and train of symptoms presented are taken into account. The use of the thermometer coupled with examination of the blood will soon establish a diagnosis. Infected horses seldom remain for more than a week without showing trypanosomes in their blood. The simplest way of examining the blood with a view to diagnosis is to place a small drop on a slide and cover it immediately with a cover-glass, applying slight pressure to cause the blood to spread out well. Such a preparation should be examined at once under a moderately high power dry lens, such as $\frac{1}{6}$-inch.

In moist blood films examined in this way trypanosomes may be easily detected if they are present even in small numbers, on account of their active movements and the disturbance they cause among the corpuscles. Trypanosomes can with a little practice be detected under much lower magnifications than that given by the $\frac{1}{6}$-inch lens. It is often advantageous to use a $\frac{2}{3}$-inch lens in conjunction with a high power eyepiece as a larger field is brought into view. This is particularly the case when trypanosomes are present in very small numbers.

Failure to find trypanosomes on any particular day or succession of days less than say a week does not warrant a negative diagnosis, but failure to find trypanosomes in the blood on ten consecutive days is almost proof that the horse is not affected.

In Camels.—The diagnosis of surra in camels presents more difficulty than in the horse, for the reasons that in the great majority of cases the onset of the disease is more insidious, its symptoms less pronounced, and its course more prolonged. Further, trypanosomes are discoverable in the blood by microscopical examination with far less frequency than in the horse. In this connection it must also be remembered that extreme familiarity with the habits, etc., of healthy camels is essential before slight departures from the normal can be detected.

In acute cases in camels diagnosis is easy, because trypanosomes are very frequently present in the blood, and daily examination for a few days will establish it. In chronic cases the periodic appearance of trypanosomes in the blood varies with the stage of the disease. In the earlier stages they may be present three or four times in a month, but as the disease progresses intervals of two or even three months may occur between paroxysms of fever during which trypanosomes are present in the blood. It is not infrequently possible to prove the presence of trypanosomes in the blood, even though they may be undiscoverable by microscopical examination, by using some of the suspected blood for the inoculation of susceptible animals, such as guinea-pigs.

In the Dog.—Microscopic examination of the blood for a few days in succession is sufficient to establish the diagnosis. In a case recorded by Gaiger trypanosomes were present on six days out of ten.

In Cattle and Buffaloes.—In India cattle and buffaloes may be infected with surra without showing any clinical evidence of the disease. Microscopical examination of the blood carried out daily for long periods may fail to reveal the presence of the trypanosomes. If, however, the temperature be taken systematically it will probably be found to show variations, and examination of the blood when the temperature is high may reveal the parasite. In some cases, however, microscopic examination of the blood may fail, and a positive diagnosis can only be based upon the results of inoculation of susceptible animals.

In India surra is not a fatal disease of cattle and buffaloes, but it may play some part in causing death when an infected animal is the subject of some intercurrent disease such as rinderpest.

In Elephants.—Not much information is available regarding surra in the elephant, but it would appear that the clinical symptoms of the disease are not very marked, and, as with the camel, great familiarity with the natural habits of the elephant is essential before slight departures from the normal can be detected.

Microscopic examination of the blood carried out systematically will generally establish a diagnosis, as trypanosomes may be swarming in the blood.

Treatment.—A vast amount of work has been done in different parts of the world in attempting to devise methods of treating surra and other types of trypanosomiasis, but it must be admitted that the results have not been commensurate with the efforts. In horses the treatment of the disease cannot even now be said to have passed out of the experimental stage. In view of the large number of investigators that have taken the subject up it is natural that an immense number of drugs should have been tried.

Experience has shown that arsenic in some form or other, alone or in conjunction with other drugs, has given the best results. But even this has not yielded uniformly good results. Variations in virulence of the trypanosome in different countries and in different parts of the same country, and variations in susceptibility of animals, have played a part in producing inconstant results.

Whatever line of treatment is adopted and in whatever form the arsenic be administered the cardinal points in the treatment are (1) that a full subtoxic dose be administered with the object of killing off the trypanosomes before they can acquire a tolerance for the drug, and (2) very careful observation of the animal under treatment, and graduation of the doses.

It is impracticable in an article of this nature to give details of the almost innumerable variations and combinations of arsenic with other drugs that have been tried, but Holmes,[1] who did a vast amount of work on this subject, found that good results were obtained by working on the following lines : A dose of atoxyl was first given to clear the circulation of trypanosomes as far as possible, and white arsenic was then given in bolus on alternate days until ten doses had been given. The doses of arsenic were gradually increased during the treatment. Extreme care was necessary in making observations on the animals so that in the event of symptoms of arsenical poisoning showing themselves the treatment could be suspended until they had passed off. This tends to make the method unsuitable for routine application. In any case, whatever method of treatment is resorted to, no horse must be considered as cured until a period of some months has elapsed without trypanosomes reappearing in the blood, during which the animal has been under close observation and systematic examinations of its blood have been made.

In camels a certain amount of success has attended efforts to effect a cure. Here, too, great care must be exercised in carrying out the treatment. The drugs that have given success are almost exclusively forms of arsenic.

In camels greater caution must be exercised in declaring an animal cured than in horses because the disease runs a far more chronic course. Camels that have been under treatment and are considered as cured must be kept under close observation for at least three months and preferably longer, with daily records of their temperatures and blood examinations, before a definite opinion can be expressed.

It is of the utmost importance in the treatment of camels that animals should be under observation and that their blood should be examined constantly for some time before treatment is begun, so that an idea may be formed as to the course the disease is taking. If this is not done the results of the treatment may be misjudged.

It is stated that arsenic combined with good nutritious food yields good results in elephants.

Up to the present no satisfactory treatment for surra in dogs has been recorded. Gaiger found that atoxyl combined with quinine failed to cure infected foxhounds.

Cattle and buffaloes very seldom come as subjects for treatment owing to the fact that they may be infected without showing symptoms.

In all animals careful nursing and freedom from work are essential parts of the treatment whatever drugs may be employed.

An animal that has been cured by means of drugs is not immune but is capable of becoming re-infected.

Immunity.—So far all efforts to produce immunity by artificial means have failed. Numerous attempts have been made to prepare both vaccines and protective sera against surra, but without success.

Prophylactic Measures.[2]—In a country like India, which is so to speak the home of surra, no prophylactic measures which can be applied on a large scale have as yet been discovered. The slaughtering of infected and in-contact animals is obviously impracticable, owing to the existence of reservoirs of the virus. Further, the disease has obtained such a hold of the country, especially in some parts such as the Punjab, that the pecuniary loss entailed by slaughter of infected and contacts would be so immense that it could not be faced, even if such methods offered a chance of stamping out the disease.

As already stated, no method of conferring immunity has been discovered up to the present. Vaccination of any kind is therefore inapplicable.

The chronic course taken by the disease in the majority of camels and the difficulties attaching to diagnosis in such cases render isolation impracticable.

Since the disease is spread by flies, and since a number of genera of flies occurring in different parts and in different types of country have been found to be capable of transmitting the disease, it is difficult to see what measures, if any, can be taken to deal with the disease through the flies.

The occurrence of the flies over wide areas, but especially upon roads and tonga routes, makes it impossible to close areas to traffic.

Numerous experiments have been made with the object of discovering some material which could be applied to the skin of animals and would have the effect of repelling the flies and preventing them from biting. In no case has any dressing been found which will prevent flies from biting for more than 15 hours or so after its application. It is therefore impracticable in the present state of knowledge to carry out prophylactic measures on anything approaching a general scale.

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[1] See also footnote on treatment of Trypanosomiasis.

[2] See also "Trypanosomiasis," Prophylaxis.

Under certain conditions something can be done to lessen the risk of infection among a given group of animals or with an individual animal. Such measures as can be taken depend upon a knowledge of the habits of the types of biting flies in the district.

Generally speaking, the flies feed in the cool of the morning and in the evening before dusk. It has also been observed that blood-sucking flies rarely if ever enter dark stables.

If the horses are out at grass during the surra season, as they frequently are in the Punjab, the risk of their becoming infected from other animals (camels) grazing with them would be reduced if they were kept in dark stables during the periods when the flies are feeding.

Differential Diagnosis.[1]—In equines in India the diagnosis of surra does not present any difficulty because trypanosomes can always be found by methodical examination of the blood for a short time, and because, so far as is known, it is the only trypanosome infection of equines occurring in the country in which invasion of the blood by the parasite in large numbers is a constant feature.

The matter presents very considerable difficulty in countries where other equine trypanosomes occur, as, for example, in Northern Africa. It cannot be said that the subject of the identification of trypanosomes is in a satisfactory state at present.

In the early days of the discovery of trypanosomes great importance was attached to details of morphology for the purpose of identification. As a matter of fact morphological details can be relied upon in certain cases for the identification of certain trypanosomes, but that is not the case with the *T. evansi*.

Subsequently a certain amount of importance was attached to the results of experimental inoculation of animals. Variations in virulence and in susceptibility tended to discount this method of identification. At a later date an effort was made to found a system of identification upon charts constructed upon the variation in length of large numbers of trypanosomes. Many variable factors, such as the stage of the disease at which the trypanosomes were measured, the actual number measured, etc., were found so to change the charts that the method had little more than a general value. The method was of more value when applied to the so-called polymorphic trypanosomes than to the monomorphic types such as the one under consideration.

Great value is placed by certain French observers upon what are known as "cross-immunity tests" for the identification of trypanosomes. These tests depend upon the specific nature of the immunity conferred upon an animal by recovery from infection with a particular trypanosome, the animal remaining susceptible to inoculation with other species of trypanosomes.

In view of the fatal nature of the infection produced in the great majority of animals by inoculation with pathogenic trypanosomes the value of this method as a routine measure is somewhat discounted.

In Africa, where numerous species of trypanosomes exist and where there are numbers of species of flies concerned in their transmission in which the trypanosomes undergo a cycle of development, a certain classification can be constructed which is based upon the parts of the fly in which these phases of development occur. This method is not applicable to surra because, as far as is known, no cycle of development occurs in the flies, the transmission being purely mechanical.

A. L. S.

MAL DE CADERAS

Mal de caderas is a disease of equines occurring in South America and is caused by a trypanosome—*Trypanosoma equinum.*

It is uncertain when the disease was first recognized, but it appears to be a fact that it was responsible for enormous losses among equines in 1860.

The disease has a wide distribution in South America, but it is mainly in the central part of that continent that it causes the most serious losses. It occurs in parts of Brazil and Bolivia, Paraguay, the Argentine, and other States. It has not been recognized in any other part of the world. Mal de caderas has a regional and seasonal distribution. It occurs on marshy land and during the period April to September, which is the dry half of the year.

A number of workers had undertaken investigations of mal de caderas prior to 1901, when the causal trypanosome was discovered by Elmassian and Migone.

The trypanosome, according to Elmassian, measures from 20 to 25 microns in length excluding the flagellum, and 2 to 3 microns in width. According to Laveran and Mesnil, the parasite is both shorter and more slender than this. Forms that are undergoing division may measure from 28 to 30 microns in length by 1 to 4 microns in width. In fresh blood the trypanosome is actively motile, but the motility is mainly of a wriggling type which does not cause much translation. According to Laveran and Mesnil, the figures given hold good for the size of the trypanosome in equines as well as in animals susceptible to experimental infection. The striking feature in connection with the morphology of this trypanosome is that in parasites stained by usual methods (*e.g.* Giemsa) the centrosome is either extremely small or apparently quite absent. Multiplication of the parasite occurs in exactly the same way as in other trypanosomes, that is to say, by longitudinal division.

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[1] See also "Trypanosomiasis," Specific Study of Species.

Vitality of the Parasite outside the Body.—Lignières has succeeded in keeping the parasites alive for three days in an ice-chest. The parasites die more rapidly at room temperatures, and if incubated death occurs very rapidly. It is said that trypanosomes retain their vitality longer in a mixture of defibrinated blood and serum than in simple defibrinated blood. According to Lignières, the trypanosomes retain their vitality longest when the serum added to the defibrinated blood is the serum of fowls, and that the other sera are less favourable to the maintenance of vitality in following order: horse and sheep, rat, ox and frog, pig, cat and dog, human serum and serum of the guinea-pig and rabbit being the least favourable. Blood containing trypanosomes retains its virulence after two hours' exposure to a temperature of −20° C., and even after exposure to a temperature of −191° C. (obtained by the use of liquid air) for five to ten minutes. The trypanosomes are far more susceptible to heat than to cold. At 40° C. blood containing trypanosomes becomes non-infective in less than six hours, while a temperature of 53° C. destroys its virulence in five minutes.

Cultivation of the Parasite.—So far no success has attended efforts to obtain and carry on cultures of the parasite. The most that has been done is to maintain the parasites living and infective in blood outside the body. Thomas and Breinl were able to prove trypanosomes to be pathogenic twenty-nine days after the inoculation of their tubes.

Animals susceptible to Infection with T. equinum.—Under natural conditions equines are practically the only animals known to suffer from mal de caderas. Elmassian records one outbreak among dogs. The disease is responsible for losses mainly among horses; mules and asses, although naturally attacked, are far more resistant.

Experimentally the disease may be transmitted to a large number of species. It is said that the period of incubation is shorter when the infective material is introduced intravenously than when it is inoculated subcutaneously. Similarly the number of trypanosomes present in the material used for inoculations influences the period of incubation and the duration of the disease. In white mice subcutaneous inoculation is followed by the appearance of trypanosomes in the blood on about the third day, and death takes place in about a week. When intraperitoneal inoculation is resorted to these periods are shortened. Grey mice are said to possess a higher degree of resistance. In these animals the trypanosomes generally increase without remissions up to the time of death. In rats the period of incubation and the duration of the disease are a day or so longer than in mice. In dogs there is usually a period of incubation of about a week, and the disease terminates fatally in about a month. The number of trypanosomes present in the blood does not increase steadily up to the time of death; there being more or less marked remissions during which trypanosomes are very scantily present. Such remissions are, as a rule, very short. In rabbits the periods of incubations and illnesses are very similar to those in dogs, but trypanosomes are never numerously present in the blood.

In the capibara, which is believed by some investigators to act as a reservoir for the virus, the period of incubation is about ten days, and the duration of the disease from one to two months. Guinea-pigs inoculated subcutaneously show trypanosomes in their blood about the ninth day. This species appears to possess some degree of resistance, as death takes place, on an average, after about three months. As in the dog, the number of trypanosomes present in the blood varies at different stages of the infection. Trypanosomes are sometimes present in large numbers, and at others not discoverable by microscopical examination. Adult cats possess a high degree of resistance, and, according to Elmassian and Migone, trypanosomes make their appearance in the blood in small numbers only. Recovery frequently takes place. Kittens inoculated with *T. equinum* usually die in from seven to eight weeks.

Cattle, sheep, and pigs inoculated with mal de caderas show practically no evidence of having become infected, but their blood can be proved to be infective by inoculation for several weeks, showing that, while incapable of multiplying to an extent sufficient to set up the disease, the trypanosome is capable of maintaining its vitality and virulence in the bodies of animals of these species for a considerable time.

Birds are absolutely immune to infection.

Methods of Infection.—As already stated, mal de caderas is easily transmitted to susceptible species by inoculation with small quantities of blood containing the trypanosomes. Ingestion of virulent blood always fails to cause infection.

The method of infection in naturally contracted cases of the disease is still a matter of doubt. It is, however, quite certain that transmission is not effected during copulation as is the case in dourine.

According to Elmassian and Migone there is no evidence to show that flies play any part in the transmission of the disease. The facts observed by these authors tend to show that they do not. This view is supported by Lignières. On the other hand, Sivari and Lecler claim to have succeeded in transmitting the disease through the medium of *Stomoxys calcitrans*, *Stomoxys nebulosa*, and unnamed *Tabanidæ*. Elmassian and Migone base their view upon facts observed under natural conditions, and Lignières' support of this view is based upon facts observed under experimental conditions. Lignières found that while healthy horses stabled with infected horses failed to contract the disease although *Stomoxys calcitrans* was present, these flies contained trypanosomes which proved infective by inoculation. It must be admitted that on *a priori* grounds it might be held that flies play an important part in the transmission of the disease, for it can be shown experimentally that it is sufficient to soil a small wound with a trace of blood containing the parasite to set up the disease.

Clinical Aspects of the Disease.—Elmassian and Migone describe three forms of mal de caderas: (1) the ordinary form, which runs a fairly *acute course*; (2) a *chronic form*; and (3) a *spasmo-paralytic form.*

As already mentioned, the disease attacks the equidæ in general, but more particularly the horse. Mules are less frequently affected than horses, and it is rare to see a case in an ass.

Acute Form of the Disease.—The disease is insidious in its onset, the first evidence of infection being rapid and progressive emaciation and depression. If the temperature be taken frequently it is generally found that there is a rise to 104° F. or even more. This may last only twenty-four hours, after which the temperature becomes normal again. With the emaciation there is also progressive anæmia. After a variable length of time evidence of paralysis in the hind legs is seen. The hindquarters sway from side to side while the animal is moving, and the hind feet are often dragged along the ground. It is to this characteristic symptom that the disease owes its name (mal de caderas—disease of the croup). The symptoms described become more and more pronounced until the animal is unable to stand without support, and animals at this stage of the disease are often seen with their legs spread out as far as possible in their efforts to maintain a standing posture. If animals in this condition are carefully looked after, death may be delayed for a few days, but in animals at all neglected death rapidly ensues, as they are unable to eat or drink. In the final stages the paralysis becomes generalized.

Elmassian and Migone have never seen a case of recovery.

Broadly speaking, an infected animal's temperature is always above normal, and it is noted that the temperature is almost invariably lower in the morning than in the evening. Charts show also marked elevations of temperature at intervals of four to five days. The rise of temperature usually occupies three or four days, while the fall is usually very sudden. Death may take place at any phase of the temperature. Trypanosomes are usually discoverable in the blood during the exacerbations and not at other times.

In the early stages of the disease the urine is distinctly abnormal; it is pigmented a deep yellow colour and has an oily appearance, and contains a considerable amount of albumen. Micturition is frequent. Later blood makes its appearance in the urine, the amount present depending upon the gravity of the changes occurring in the kidneys. The urine in less severe cases is merely flecked with blood, but in severe cases its appearance may closely resemble blood itself. Elmassian has never found trypanosomes in such urine.

In some cases lesions develop in connection with the skin. These lesions take two forms. Raised areas measuring an inch or two in diameter make their appearance. Frequently the hair is shed over these patches, and in such cases it can be seen that the skin is covered with thin crusts of dried exudate. If these be removed it is found that the skin is inflamed and shows a number of cracks or fissures from which the exudate escapes. These lesions are most frequently found on the neck and loins. The second form of skin lesion is a temporary subcutaneous infiltration developing usually on the legs in the neighbourhood of joints. Œdema of the eyelids accompanied by muco-purulent conjunctivitis is a frequent symptom. This is sometimes associated with interstitial keratitis. The cornea, especially the lower part, shows irregular milky patches. These, however, disappear without leaving any permanent injury. There is little evidence of disturbance of respiration except during the periods of high fever and during the period immediately preceding death. Digestive troubles are not marked, but as the paralysis, which is first observed in the hind legs, extends, the sphincters of the anus and bladder become involved, permitting the frequent involuntary expulsion of fæces and urine. The anæmia which is a pronounced lesion of the disease is evidenced by acceleration of the pulse, especially during the febrile periods, and pallor of the visible mucous membranes.

Appetite is not lost during the whole course of the disease, but animals are usually incapable of feeding for a short time prior to death.

Chronic Form of Mal de Caderas.—According to Elmassian, stock-owners in areas where this disease occurs consider it to be a condition entirely distinct from true mal de caderas, but they admit that an animal which in the first instance is attacked by this disease, which they term Baacey-poy or Baccy-poucou, invariably dies of mal de caderas. In the course of his investigations Elmassian encountered a number of cases of this type, and in every instance was able to prove the existence of the trypanosome of mal de caderas in the blood. This chronic form of the disease generally occurs as an epizoötic, but occasionally isolated cases occur, and sometimes cases are observed in the course of an outbreak of the ordinary or acute form of the disease.

The *symptoms* in this form of the disease are in their sum identical with those seen in the more acute form, but their development is far slower. It is noted, however, that although there is fever with remissions the temperature does not rise so high as in the acute form. Further, after a time even this fever disappears. Trypanosomes are not discoverable in the blood by microscopic examination, but their presence there can be proved by the experimental inoculation of susceptible animals. It is not known why in some cases the disease assumes the chronic form. Elmassian states that in horses which he inoculated with blood from a horse suffering from the chronic form of the disease, the ordinary acute form developed with the utmost regularity and certainty, trypanosomes making their appearance in the blood after the interval of a week.

On the other hand, if trypanosomes are passed through a series of somewhat insusceptible animals such as cats, prior to the inoculation of a horse, the horse develops the chronic form of the disease.

The Spasmo-paralytic Form of the Disease.—Only two cases of this form of the disease appear to have been observed by Elmassian. It is to be noted, however, that in these cases the disease did not appear to be a pure trypanosome infection, a second parasite of a different nature being present in the blood.

One of the affected animals was kept under close observation. The first symptom noticed was that the animal did not feed well. Close examination showed that there was slight spasmodic contraction of the muscles of mastication. This was not always present, so that the animal was able to eat and drink. Emaciation, however, became more and more marked. When the animal had been under observation about four days it was seized with a kind of epileptiform attack involving the muscles of the head and neck, which resulted in the head being drawn round to the left side. The attack lasted for about ten minutes, and during that period the spasms varied in intensity in different muscles and groups of muscles. During the following twenty-four hours four or five of these attacks were observed. The animal remained subject to attacks of this kind for about ten days, after which all symptoms of a nervous nature ceased. From this date, however, mal de caderas developed and ran a typical course, death occurring sixteen days later. Daily examination of the blood failed to reveal any trypanosomes until the fifth day after the animal first came under close observation. On this occasion their presence was proved by the inoculation of a susceptible monkey. The course of the infection in the monkey showed that the trypanosome was of a lower degree of virulence than normal.

Although examination of the blood had failed to reveal the presence of trypanosomes, yet there were always present numbers of another type of parasite. These were long ribbon-like structures measuring about 60 microns in length by 8 to 10 microns in width. They were pointed on one end and rounded or square at the other. They contained five or six nuclei, which varied in size and were disposed in line in the centre of the body, the larger ones being placed towards the thick end of the parasite and the small ones towards the pointed end. In addition to these, there were small rounded or square forms with a single large nucleus and a small amount of cell body. Elmassian considers two possibilities with regard to the presence of this exceptional type of parasite in a case of mal de caderas. First, that there was actually a double infection. In all animals inoculated with blood from this horse the ordinary form of mal de caderas developed, the second type of parasite failing to appear in the blood. The spasmo-paralytic symptoms also failed to develop. The second possibility is that the second type of parasite was present in the blood without being responsible or in any way connected with the symptoms produced. In this case the whole train of symptoms observed would appear to have been caused by the trypanosomes.

The Reservoir of the Virus.—It has been suggested that the capibara is the source of the virus in outbreaks of the disease among horses. It is said that actual outbreaks of the disease occur among these animals, which are very common in the areas where it occurs among horses. Migone states that he has had the opportunity of examining a number of capibaras in the course of an outbreak, and that he proved the presence of trypanosomes in their blood (1910). Neira, writing in 1913, states that he does not find any correspondence between the distribution of the capibara and mal de caderas. He states that he has been able to infect the vinchuca experimentally and, further, that the fæces from such infected animals were capable of infecting guinea-pigs when placed on the cornea of the eye.

Diagnosis. — In the ordinary form of the disease diagnosis presents no difficulty. The following points are of value in arriving at an opinion: (1) the known distribution of the disease; (2) the constant train of clinical symptoms; and (3) microscopic examination of the blood for trypanosomes, the centrosome of which is either exceedingly small or absent.

In chronic cases diagnosis presents greater difficulties. In the first place the symptoms are not at all characteristic until the late stages are reached. In the early stages emaciation is practically the only evidence of disease. Further, frequent examination of the blood may fail to reveal the parasite. In such cases resort must be had to the experimental inoculation of animals. In order that a large dose of blood (20-30 c.c.) may be injected, a horse should be selected for the purpose.

Treatment.— Some good results have been obtained in the treatment of mal de caderas in laboratory animals with a variety of drugs, but up to the present no drug has been found which will effect a cure with certainty in horses. In some cases arsenious oxide has given favourable results, but in others its administration has resulted only in a temporary disappearance of trypanosomes from the blood. According to Laveran and Mesnil the sera of sheep, goats, and cattle that have been infected with the disease possesses some immunizing properties for a short time after the animals have recovered from the infection. This power is, however, of short duration only.

Prophylaxis.—In view of the absence of any exact knowledge regarding the natural method of transmission of the infection and the reservoir of the virus, it is impossible to frame any definite rules for the control of the disease, and little more can be done than to put into force the general principles governing the control of contagious diseases: (1) isolation or destruction of diseased animals; (2) careful and repeated examination of the apparently healthy with a view to arriving at an early diagnosis when fresh cases occur.

In view of the fact that the disease is most common on marshy land and along the banks of rivers, such places should be avoided. The possibility that the capibara acts as the reservoir for the virus of this disease indicates that these animals should be destroyed in as large numbers as possible.

It has been advised that stables should be provided with fly-proof wire netting; while the natural method of infection remains unknown, the value of this precaution is a matter of doubt.

A. L. S.

DOURINE

Synonyms.—Covering Disease; Breeding Paralysis; Mal du Coït (*Fr.*)**; Beschälseuche** (*Ger.*)**; etc.**

Dourine is a disease of horses and asses, and is a specific, contagious, chronic, and insidious malady caused by a protozoan parasite named *Trypanosoma equiperdum.* Diseases caused by trypanosomes have their habitat, for the most part, in tropical and subtropical countries, and depend for their propagation and transmission upon some particular insect, intermediary host, or carrier. Dourine is a unique exception, for it occurs not only in Africa and in Asia but also in many parts of Europe and America, in countries far removed from tropical conditions; further, it does not require an intermediary host or carrier but is directly communicated in nature from the diseased to the healthy animal through the act of sexual union, and, so far as is definitely known, in no other way. It is the only known trypanosome disease occurring in Europe and in North America.

History.—The disease has been known to the Arabs for centuries, it is said. In Europe it was first observed by Ammon, in Prussia, 1796-99. Outbreaks were reported from different European countries at various periods during the nineteenth century. The trypanosome nature of the disease was first suspected by Rouget, in 1894, on finding the parasite in the blood of a stallion at Constantine. Schneider and Buffard, in 1899, found the trypanosome in two dourined horses in Algeria, and were the first to experimentally reproduce the disease by inoculations of the parasite. On account of the many failures to find the trypanosome in different outbreaks of dourine in different countries, doubts were sometimes raised as to the etiological importance of this parasite. However, these doubts have all been set aside and the causal rôle of the trypanosome fully confirmed by the rediscovery and study of the parasite in Hungary (Marek), in Prussia (Meissner), in Canada (Watson), and in the United States (Mohler).

Geographical Distribution.—Probably of tropical origin, the disease has been carried to various parts of the world through the transportation of infected stallions, mares, and donkeys. Once introduced the disease is apt to become largely scattered by means of infective coitus and breeding operations, and owing to its insidious character is most difficult to suppress. The disease has been reported from nearly all the countries in Europe. It exists in Northern Africa, and along the southern littoral of the Mediterranean; in South Africa, India, and Western Asia; in Canada, in the United States, and in several of the South American republics.

Pathogenic Agent: *Trypanosoma equiperdum.* Doflein, 1901.

The dourine trypanosome is a flagellated protozoan parasite closely resembling other pathogenic trypanosomes, notably those of surra, nagana, mal de caderas, and sleeping sickness of man, in morphology, staining reactions, vitality, and general biological features. The posterior extremity of the dourine trypanosome has often a cut-off, blunted, or fish-mouth appearance which, in stained specimens, may aid in distinguishing it from other trypanosomes. A very common feature is the presence of a vacuole bordering upon the centrosome. The Giemsa method of staining is the best for demonstration purposes. It gives a very beautiful picture of the parasite, colouring the nucleus purple violet, the centrosome black, cytoplasm a light greyish blue, metachromatic granules dark blue, undulating membrane pale pink, and flagellum red.

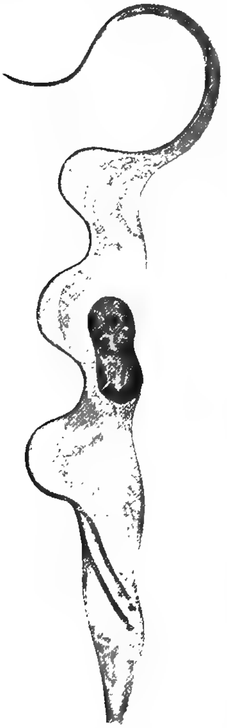


FIG. 34.—Trypanosome of dourine. (*T. equiperdum.* × 4000.)

The detection of the trypanosome in horses is usually very difficult and may involve a prolonged search extending over many weeks or months, which even then may prove unsuccessful. For a long time it was thought that the dourine of Central Europe and of America was a different entity from that of Algeria and tropical countries, for the reason that in the latter the trypanosome could usually be found by direct examination or by inoculation experiments, while in the former all attempts to demonstrate the presence of the parasite were for many years entirely unavailing. However, the single nature of dourine is no longer questioned, but it happens that in certain countries outside of the tropics the parasite modifies its habits, and in equines does not appear as a true blood parasite but rather as a tissue parasite and extraordinarily difficult to locate. It gains an entry in the first place by way of the genital tract, and develops in the mucous membranes and submucous tissues of the urethra in stallions and of the vulva and vagina in mares, where it causes a more or less fugitive infiltration and œdema. Later on, the parasites multiply under the skin in various parts of the body and give rise to small flattened œdematous patches or plaques which come, as a rule, singly and but rarely, quickly vanishing and easily escaping observation. It is these œdemas, whenever and wherever they occur, that should be examined and thoroughly searched, and the less blood there is present in the specimen obtained the better the opportunity of finding the parasite.

Natural Dourine Infection of Horses

Evolution and Symptoms of the Disease.—The starting-point of infection lies in the genital tract at the time of infective coitus. From that given time and place the course and manner of evolution of the disease is irregular and uncertain. An acute case of natural dourine is extremely rare and is hardly ever to be observed in practice. The disease is essentially a chronic and intermittent one. The determining factors in its development are (1) the degree of virulence of the race of infecting trypanosomes; (2) the varying degree of resistance on the one hand, and of high susceptibility on the other hand, of different breeds and types of horses; (3) climatic conditions; and (4) physical and functional conditions dependent upon stabling and exhausting work, breeding, and a more or less natural life on the open range or pasture. As a rule, tropical strains of dourine possess a higher degree of virulence than that of the strains propagated in temperate zones, and, correspondingly, the development of the disease is less obscure and the symptoms more pronounced. The higher breeds of horses are the most susceptible. In animals of the thoroughbred type the disease is apt to make quicker progress and manifest itself mainly in derangements of the central nervous system. In the more sluggish breeds, such as the Clydesdale type, the disease is more halting and is marked by œdemas and disturbances of the circulatory system. Native breeds of ponies are the most resistant, and frequently serve as carriers and reservoirs of the virus for a duration of years. Donkeys also are well able to tolerate infection and become dangerous carriers. Climatic changes and extremes of temperature serve either to arrest or advance the progress of disease. Thus, in Canada, for example, the disease often lies dormant through the winter months and with the first hot spell of weather in June or July becomes suddenly and increasingly active. Work, breeding, and physical exhaustion favour the disease, especially in the stallion. Mares that have become impregnated at the time of the infective coitus occasionally abort; but more often they carry their young to full term. It has been noted again and again that pregnant mares have remained apparently healthy up to the time of parturition, and that very shortly after the event and about the time of the first period of œstrum there occurs a rapid change from a healthy to a diseased condition in which unmistakable symptoms of dourine are presented. Under natural conditions on the open ranges or pastures more resistance is shown than in stable-kept animals.

Period of Incubation.—The first symptoms may appear in as early as two weeks from the day of the infective coitus or as late as two or three months. In not a few cases, and particularly in cold countries, no symptoms can be recognized until the first winter has passed and the second spring or summer arrives. But if the period of incubation is regarded from an immunological aspect and as a period of sensitization, then it is less than one month, and specific serum reactions can usually be obtained in from ten to twenty days after infection.

Period of Disease.—This is rarely under six months; it averages between one and two years; frequently it extends to three and sometimes to five years. As regards infection and immunity reactions the period may extend to over ten years, several cases having been observed for over a ten-year period at the Government Dourine Research Station in Canada.

In most accounts of dourine the evolution of the disease is divided into three well-defined periods or stages, and the symptoms grouped to correspond, thus: (1) *Primary*, œdema, tumefactions and changes in the genital organs; (2) *Secondary*, plaques and skin eruptions; (3) *Tertiary*, paralysis, anæmia, and cachexia. This is an arbitrary arrangement, convenient for descriptive purposes but rather apt to mislead the inexperienced observer of this disease in his conclusions or diagnosis. Actually, the disease operates in a succession of attacks, each attack being followed by a period of tolerance or immunity for which no time-duration can be fixed or even indicated.

One attack may follow another so closely that symptoms are never altogether absent. On the other hand, the attacks may be so far apart that in long intervals between them there is no visible sign of disease. Unless such an interval has continued for at least two years without a relapse it would be unsafe to assume that the animal had permanently recovered. As for the grouping and sequence of symptoms, it should be remembered that symptoms of all three groups may be coexistent; or, those of one group may appear simultaneously with those of another; and that symptoms grouped as "primary" may recur after the first, second, third, fourth, and any subsequent attack, and, similarly, those of the "secondary" or "tertiary" groups may recur not once but repeatedly and irrespective of the sequence or order indicated.

The period of disease has its different stages of exacerbation, tolerance, relapse and, finally, recovery or decline. Any of these stages may be of short or of long duration. They may occur but once or recur again and again. The symptoms most frequently met with are fever, tumefactions and local œdemas of the genital parts and mammary glands, œdematous eruptions of the skin, knuckling of the joints and loss of co-ordination, facial paralysis and ocular troubles, anæmia and emaciation. The only truly pathognomonic symptom is the œdematous patch or "plaque." Unfortunately, for diagnostic purposes, the "plaques" are few and far between; they appear suddenly and vanish within a few days, and in many cases never occur at all, or at least are never observed. In the early stages of the disease fugitive œdemas of the genital organs, and febrile paroxysms, occur as a rule.

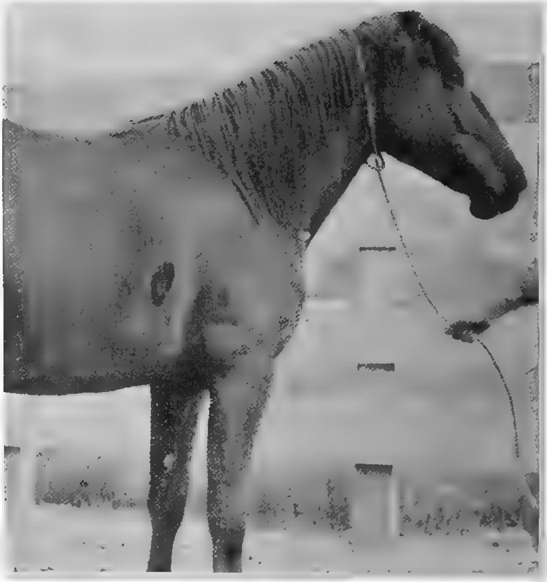
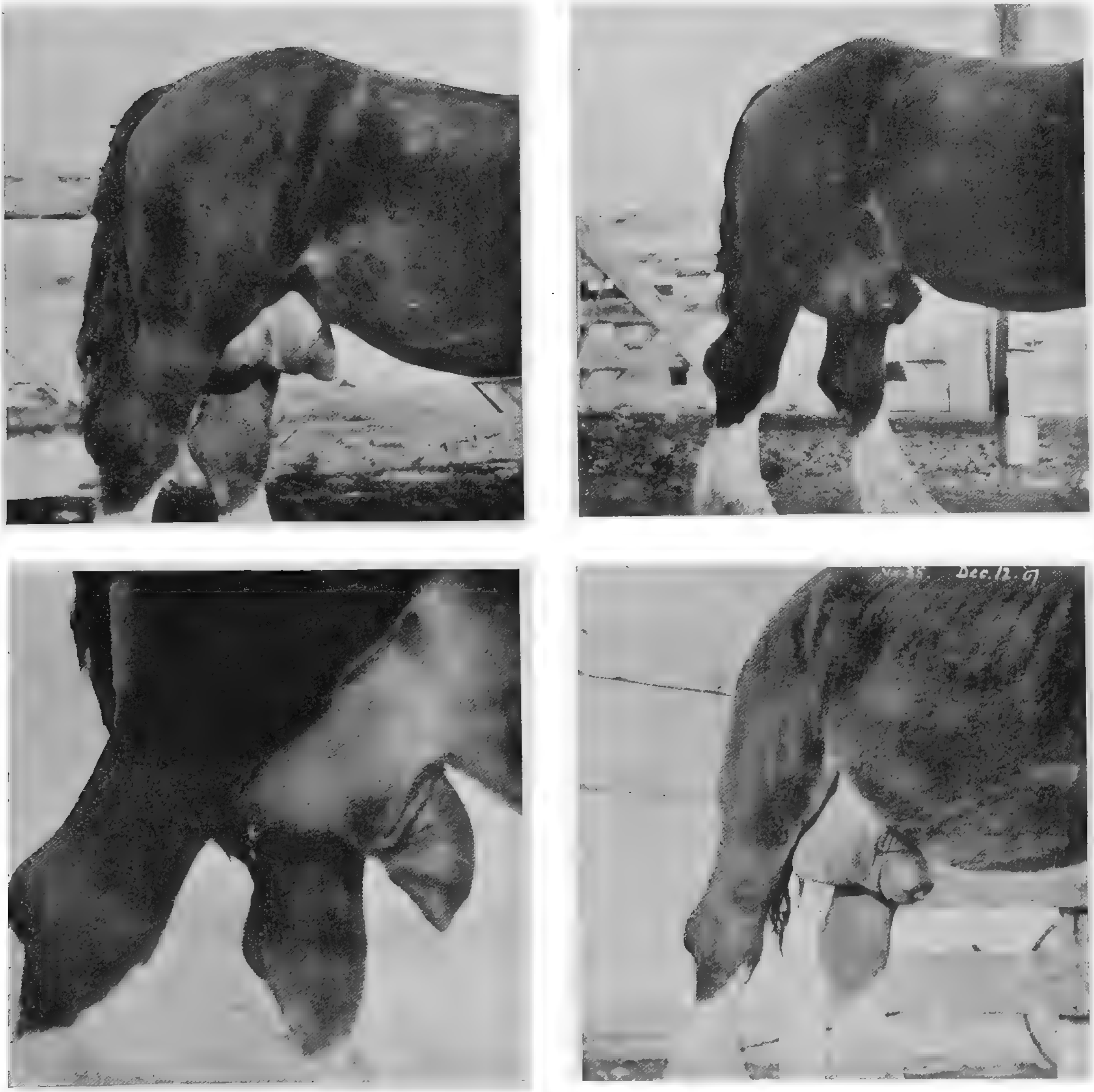


Fig. 35.—A typical dourine "plaque" occurring ten months after experimental infection. Trypanosomes present in the serous fluid.

In a **stallion** the first symptom is an œdema of the sheath, and this is often accompanied by a slight swelling of the glans penis. The mucous membrane at the urethral exit may show a bulging, infiltrated appearance and may even protrude from the opening. This first œdema vanishes in a few days and the parts appear normal again. After an interval of, usually, fifteen to twenty days, the œdema returns, and it is apt to be more pronounced. It may involve a portion of the scrotum, but it is still more or less transient, disappearing and recurring after another interval. At each recurrence the œdema tends to increase, and sooner or later the affected parts become permanently thickened and indurated. The penis may be in a state of paraphymosis, temporary or permanent. In stallions of the heavy, sluggish breeds the œdema may extend over the whole of the floor of the abdomen and dominate the clinical picture. In the lighter breeds, however, this symptom may be only slight and quite transient.

In a **mare** the first symptom is a patch of slightly swollen mucous membrane of the vulva or vagina, and is followed by œdema of the vulva. Sometimes only one labium is involved, giving the vulva a distorted appearance. The œdema vanishes and returns, the intervals becoming more and more irregular. The outer black skin of the vulva may present irregularly-shaped patches of depigmentation. The patches at first appear pink, then white. They are of uncertain duration, in some cases persisting throughout the disease, in others returning to a normal appearance. At this stage the vaginal mucosa may show raised or thickened, semi-



Figs. 36, 37, 38, and 39.—A naturally infected stallion during the 16th to 18th month of the disease.

transparent patches, and is sometimes seen to protrude into the vulva opening. The clitoris may appear swollen and erect. Frequently there is increased sexual desire, spasmodic attempts to urinate, and prolonged periods of œstrum. Apart from these conditions there does not appear any unusual vaginal discharge; but in the later stages of the disease, especially after an infected pregnant mare has aborted or foaled, a chronic vesico-uterine discharge may occur.

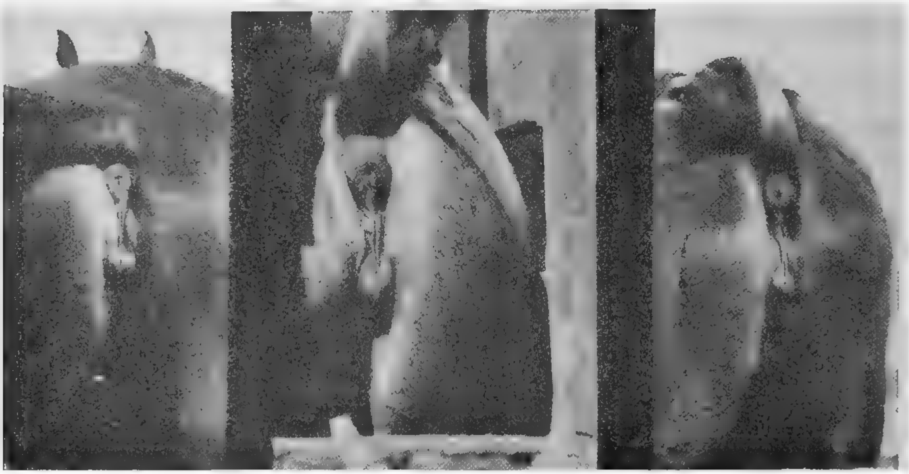


Fig. 40. Fig. 41. Fig. 42.

Figs 40, 41, and 42.—Œdematous swellings in young fillies experimentally infected. Œdema of the lips of the vulva (Fig. 40), of the anus (Fig. 41), and patchy œdema of the skin extending down between the thighs (Fig. 42). Trypanosomes present.

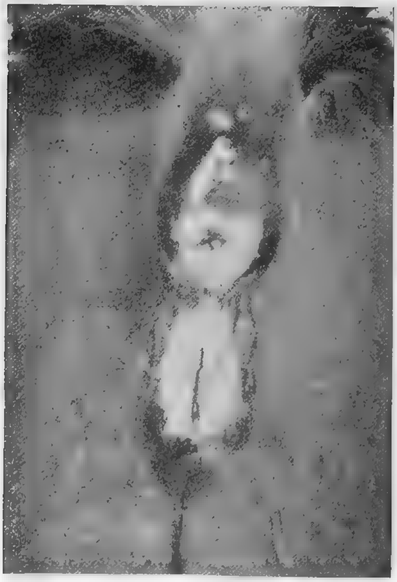


Fig 43.—Depigmentation of the skin of the vulva and anus in a naturally infected mare. Trypanosomes present in the vagina.

In mares the development of the disease is, as a rule, slower and more irregular and uncertain than in stallions. In the former the genital symptoms may be so slight as to escape notice. After infection has existed for six months or longer, and when it may be impossible to detect any abnormal condition of the genitals, it is not uncommon to find a slight œdema of

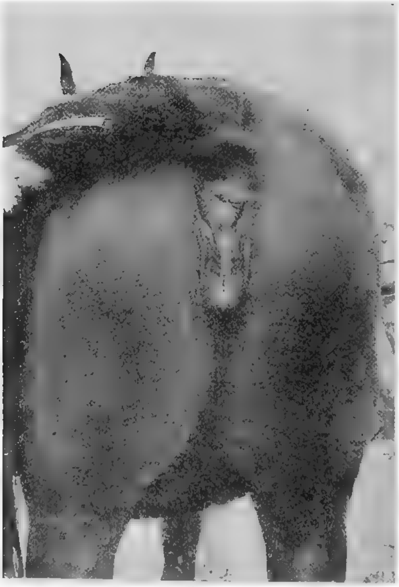


Fig 44.—The same mare ten months later. Trypanosomes absent.

the udder or of the skin and underlying tissues just anterior to the udder. Less commonly there may be noted œdema of the anus, or a patch below the vulva, or traces extending down the inner borders of the thighs. Such symptoms, slight and vague as they may appear, are of great value, for it is in these serosities, especially on the first days of their appearance, that the trypanosomes of dourine may be found by careful searching.

The true " plaque " or " dollar spots " differ somewhat in appearance from the œdematous patches already described. The " plaque " may appear upon any part of the body surface, but seems to favour the sides covering the ribs. It is aptly described as resembling " a thin disc of metal slipped under the skin." In tropical countries this symptom is said to appear at about the third month of the disease, or even earlier, the plaques tending to come in successive crops. However, even in the tropics, this would appear to be the exception rather than the rule, and in America, certainly, it is very rarely found so. In some animals a plaque has never been known to occur, in others one or several may appear at any time in the first, second, or third year of the disease, and, in rare cases, a rapid succession of plaques takes place during one brief period in the course of the disease. The duration of a plaque averages four to five days, ten days being the maximum and one day the minimum period. In the plaques, as in the local œdemas of the genital organs, the trypanosome goes through a cycle of development which is usually completed within three days. The trypanosomes then agglutinate and are rapidly taken up by the phagocytes. It is thus important to examine these serous fluids for trypanosomes in the early stage of eruption.

The dourine *fever* may occur at any stage or period of the disease, but is most pronounced and more frequently noted in the very early stage. It is then distinctly of the intermittent or remittent type. A typical paroxysm has an average duration of five to six days, the maximum temperature, usually 104° to 105° F., being reached about the third or fourth day. The intermission ranges between twenty-one and twenty-eight days. The febrile stages are closely associated with the development of successive crops of trypanosomes. A renewal of the genital symptoms, the eruption of a plaque, or a recurrence of œdema on some part of the animal body can usually be noted during or just preceding the rise in temperature. As the disease makes progress, or as a tolerance to it is acquired, febrile conditions are variable and inconstant. In some cases, towards the later stages of the disease, a subfebrile temperature of about 102° F. may continue for long periods.

The disturbance of the *nervous system* is shown by inco-ordinate locomotory action, and paraplegic conditions of the hind limbs, lips, nostrils, ears, and throat. The animal has a " wabbling " gait, swaying from side to side; there is knuckling over at the fetlock-joints, and, in unshod animals, the toes of the hind hoofs wear down from dragging and shuffling the feet. Facial paralysis is nearly always unilateral. The ear, upper eyelid, nostril, and under lip may be affected at the same time, or only one of these appendages. Such symptoms are not as fugitive as those of the other groups, and tend to persist.

Ocular symptoms are not constant and occur in only a small percentage of cases. They comprise photophobia, lachrymation, keratitis, and corneal opacities and changes in the interior of the eye. Stallions seem more prone to ocular lesions than do mares. It is of interest to note that the corneal opacity seen in the stallion which figures in the accompanying illustration (Fig. 36) was noted before any other symptoms of the disease had been observed. A number of similar cases are also on record.

In cases advancing towards a fatal termination the decline is usually slow and gradual. The appetite remains good almost to the end and often appears excessive. There is a progressive anæmia and emaciation; the animal has a most dejected appearance and continues a miserable existence until, finally, from exhaustion and paraplegia, it is down and unable to rise; unless destroyed it suffers a lingering death.

The *mortality* in tropical countries appears to be very high. In some areas, and according to some authorities, dourine is an almost invariably fatal disease. However, the hill-bred pony of Northern India, the donkey and mountain pony of the Pyrenees, and the native breeds of the prairies and western foot-hills of North America are known to be very resistant; and in such the mortality may be as low or lower than 10 per cent. In effect, mortality varies according to virulence of the pathogenic agent, the type or breed of the animal infected, and climatic conditions generally. In Canada, the average mortality would probably be less than 50 per cent. The female is more resistant than the male. In stallions the disease appears to result fatally, without exception.

Pathological Anatomy.—The principal lesions are to be found in the nervous system and are most marked in the lumbar and sacral regions. In animals in which paralytic conditions in the hind limbs have been of long duration the spinal cord shows softened, pulpy, discoloured areas. Histological examinations made by Marek showed a degeneration of the nerve-fibres of the posterior columns, and in the extra-spinal nerve-trunks, particularly of the hind limbs, a cellular infiltration, degeneration and atrophy. Mott, comparatively studying the changes in the nervous system in dourine and in infections with *Tr. gambiense*—sleeping sickness—found evidence of a marked proliferation and hypertrophy of the subpial and septal neuroglia. In the genital organs there is evidence of chronic inflammatory changes. In the stallion the scrotum, sheath, and testicular coverings are much thickened and infiltrated. In some cases the testicles are embedded in a tough mass of new sclerotic tissue and can be separated and dissected out only with much difficulty. In the mare the vulva, the walls of the vagina, the uterus and udder show thickening and gelatinous infiltration. In such cases the neighbouring lymphatic glands are hypertrophied and softened, and sometimes hæmorrhagic. Other lesions are similar in character to those found in other chronic anæmias and wasting diseases, such as gelatinous infiltrations under the skin and of the connective tissues, serous transudations and fibrinous deposits in the large body cavities, lesions in the kidneys and bladder, bone-marrow changes, etc. In dourine the liver and spleen are not enlarged and are sometimes below normal weight and size.

Experimental Dourine

Tr. equiperdum is always pathogenic for the horse. If introduced into the vaginal tract of a healthy mare it reproduces the disease in a similar fashion to that which follows an infective coitus. A subcutaneous inoculation of infective material, obtained from a plaque, œdematous swelling, or the vaginal secretions, and known to contain trypanosomes, is followed in about ten days' time by a small swelling at the seat of inoculation containing serous fluid and numerous trypanosomes. This primary swelling disappears in a few days and the disease evolves as in a case of natural infection, especially if the inoculation has been made under the skin of the vulva.

Blood drawn from the general circulation of dourined horses rarely contains trypanosomes and thus rarely reproduces the disease when inoculated into healthy subjects. Direct transfusions of blood and inoculations of large quantities repeated at intervals more often than otherwise fail to set up an infection. Only during a very active stage of the disease, as during a period of plaque eruptions, is an inoculation of blood likely to be successful.

The virulence of a strain of dourine can be raised for a given species of animal by rapid passages through a series of young animals of that species. By such means the disease is rendered more acute in foals or yearling horses, running its course in about six weeks in males and in about three months in females, a high remittent fever being the dominant feature throughout.

Very great difficulty is experienced in transmitting dourine from the horse to laboratory animals. The whole of the difficulty is in making the first passage. The white mouse seems the least resistant of the small animals; even so, it may be necessary to inoculate series after series of mice before meeting with a single successful inoculation. But once this is accomplished the trypanosome can be passed through rats, rabbits, guinea-pigs, dogs, cats, etc., with great ease, and from an animal of one species to that of another, and carried on indefinitely.

The first passage through a laboratory animal raises the virulence of the trypanosome for horses to a very high degree. In horses infected with trypanosome blood taken from a rat or guinea-pig the disease makes rapid and continuous progress. There is scarcely any remission of symptoms; trypanosomes appear early in the general circulation, and increase in number up to the time of death, which takes place, usually, in eight or ten weeks or less, in adult horses.

In mice and rats dourine infection is always acute. There is but one cycle of trypanosome development, which is usually completed in from four to six days (corresponding very closely in duration to one of the trypanosome cycles as observed in naturally infected horses), when death occurs almost without any warning symptoms. In guinea-pigs remissions occur in which the trypanosomes present in the circulating blood are much reduced in numbers. The duration of infection averages six to eight weeks. In rabbits and dogs the disease is still more remittent and chronic and has a duration of one to three months. Œdemas and ocular lesions are prominent features and, in the last weeks of the disease, wasting of the body tissues and paralytic conditions.

In some countries, and with certain strains of dourine, notably with Schneider and Buffard's Algerian strain, dogs were found more susceptible to direct inoculation from the horse than were other species of small animals. It has even been recommended to employ dogs as test animals for the diagnosis of the disease. This may be possible in some countries, but it has signally failed in Central Europe and in North America with dourine strains of, probably, lesser virulence.

As stated by Laveran and Mesnil, "The trypanosome of dourine constitutes one of the best examples of variations in the virulence of the same species following its origin and genealogy."[1]

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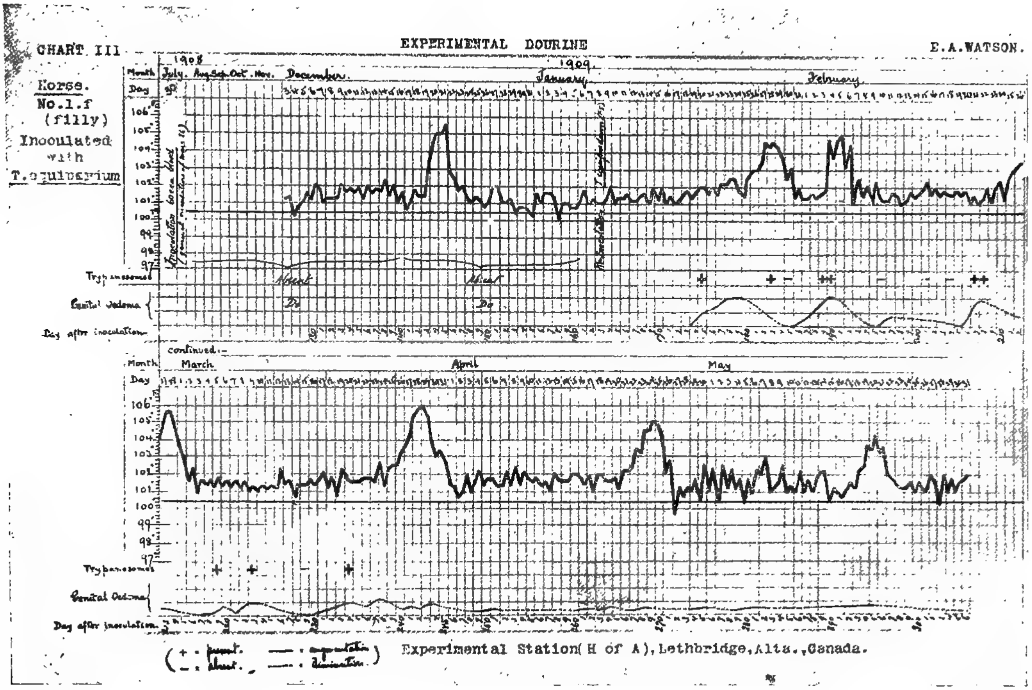
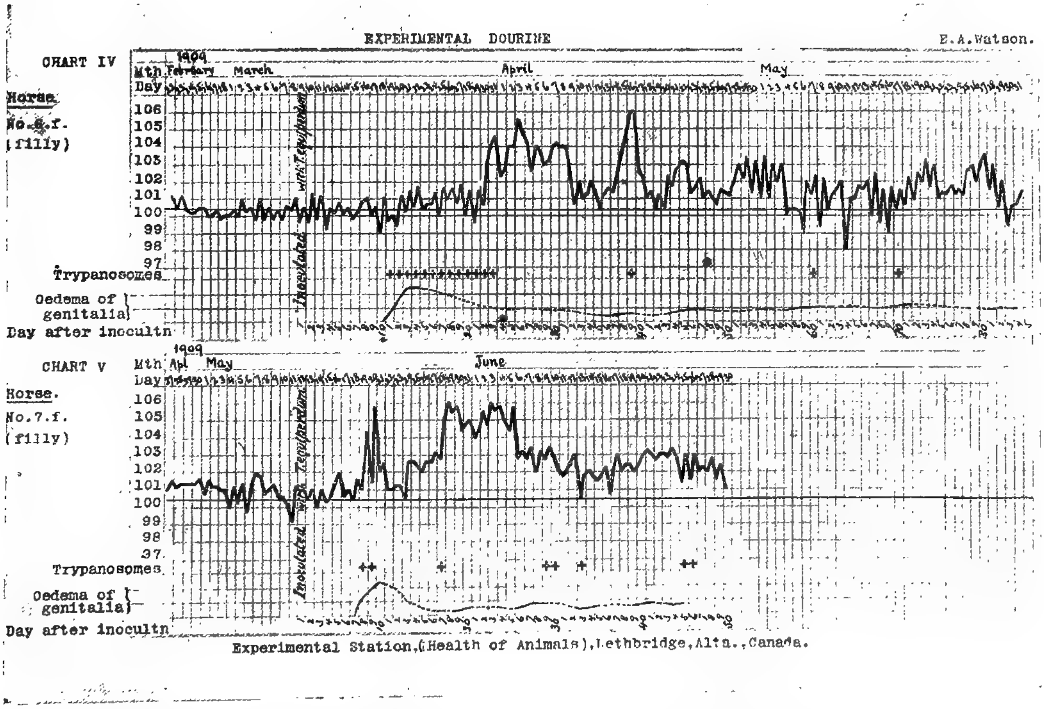
[1] Laveran and Mesnil, *Trypanosomes and Trypanosomiases*, Paris, 1912. This standard work should be consulted by all students of trypanosome diseases.





FIGS. 45 AND 46.—(Chart III., Foal No. 1; Chart IV., Foal No. 6; Chart V., Foal No. 7.) Types of dourine fever in three of a series of horse foals experimentally infected with *Tr. equiperdum* of which the virulence was raised by the serial passage. (From photographs.)

Diagnosis

Symptomatic Diagnosis.—This is based mainly upon the type of œdema affecting the genital organs, the cutaneous plaques, and the nervous symptoms shown in the hind limbs. In the early stages of the disease it is difficult and frequently impossible to detect such symptoms or to recognize them with any degree of certainty. It is always advisable to search into the history of the case or cases and to examine as many as possible of the contact animals. When it is found that only stallions and brood mares are affected there are but two covering diseases to consider, namely, dourine and coital exanthema. The latter is the disease most commonly confused with dourine, inexcusably so, for the symptoms are well defined and differ profoundly from those of dourine both in character and manner of development. The vesicles and ulcers of coital exanthema have no analogy in dourine, though statements to the contrary are made in some of the earlier works and are occasionally repeated in some of the later descriptions of dourine. It is extremely doubtful if an ulcer ever results from dourine or from any other trypanosome infection. The ulcer which very rarely is noted in a case of dourine is similar in appearance to the chronic ulcer which occurs just as rarely in stallions and mares unassociated with either dourine or coital exanthema. It has the appearance often of a canker, and is usually placed near the edges of the vulva or on the glans penis. Microscopical examination of deep scrapings from such lesions show frequently a spirochætæ infection.

Detection of the Parasite.—In Europe and in North America dourine is the only known trypanosome disease of horses and donkeys. The detection of trypanosomes in the serous fluids of the œdemas or plaques, or in the vaginal and urethral secretions, is then sufficient to confirm a diagnosis; but, unfortunately, detection is difficult, as the parasites are usually very scanty in number and for long periods in the disease are inactive and not to be found.

Serological Methods of Diagnosis.—The serum of dourine-infected animals possesses certain specific properties which can be determined by (1) the complement-fixation test; (2) the agglutination test; and (3) the precipitin test. Of these, the complement-fixation test has come to be recognized as a delicate, safe, and eminently satisfactory method of diagnosing dourine. It has been adopted by the chief veterinarians of the Canadian and United States Governments, and is applied in all outbreaks of the disease, with the result that dourine has been reduced to a minimum and almost eradicated from the country.

The serum diagnosis of dourine is essentially a test for the laboratory, necessitating the careful preparation of a number of reagents, including a trypanosome antigen, and a most exact technique throughout.[1]

Treatment

Curative Treatment.—Nearly all methods of drug-treatment are based upon the employment of arsenic. A course of treatment with arsenious acid or with cacodylate of soda often results in a marked improvement and sometimes, apparently, in a cure. Stronger claims have been made for atoxyl and for arsenophenylglycin; the latter, probably, has a greater efficacy in dourine infections than has any other preparation of arsenic. A single full dose of arsenophenylglycin will usually clear the blood of trypanosomes within twenty-four hours and cause a remission of symptoms and a remarkably rapid improvement in general condition and appearance. However, with any form of treatment relapses are apt to occur, and in such cases further treatment with the same drug is of little avail. The drug should be given in the maximum dose that can be tolerated, intravenously, and repeated in fifteen days and again after a second interval of fifteen days. Some horses will tolerate a dosage of 60 milligrammes of the drug per kilogramme of body weight; others will not tolerate more than 50 milligrammes per kilogramme.

However good the immediate results of drug treatment appear to be, a year or two must be allowed for a possible relapse and before one can pronounce on the completeness or permanency of a cure.

The disease is so insidious in character and of such a menace to the horse-breeding industry that, in general, curative treatment does not appear to be justified, except in such countries where the disease is indigenous and where eradication by "stamping-out" methods does not appear practicable.

Preventive Treatment.—The serum of animals chronically infected, and of animals which have made a natural recovery from the disease, affords a certain degree of protection to healthy animals against experimental infection. Such immunization, however, is at present mainly of experimental interest and has no practical application in the prevention of the disease.

Sanitary Police Measures

The measures for suppressing an outbreak of dourine are based mainly on (1) the stoppage of further breeding of infected animals and of animals which may have been in contact with a diseased animal; (2) the earliest possible detection of all cases of infection; and (3) the slaughter of all infected animals.

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[1] Winkler and Wyschelessky, *Berlin. Tierarztl. Woch.*, Dec. 1911; Watson, *Proc. Amer. Vet. Med. Assoc.*, 1912; *Ann. Reports Vet. Dir.-Gen. Canada*, 1912, 1913, 1914, and 1915; *Parasitology*, vol. viii. No. 2, Sept. 1915; Mohler, Eichhorn, and Buck, *Jour. Agric. Research*, vol. i. No 2, Nov. 1913.

The order to cease breeding should be given immediately an outbreak of the disease occurs, and continued in force until such time as all of the animals exposed to infection have been properly dealt with and the danger of further spread of the disease has been fully guarded against. The question of diagnosis is one of the greatest importance and of the greatest difficulty. The majority of outbreaks of dourine are due, not to reimportations of infected animals, but to some unknown carriers of the disease which have escaped detection in a previous outbreak. The fact that animals may continue for periods of one to three years in normal health and appearance while carrying a dourine infection and capable of transmitting it, brings the question of an early and definite diagnosis to the leading position in measures of control. The problem of detection is solved by the application of serum methods of diagnosis, particularly the complement-fixation test. The systematic application of this test to every animal exposed to infection rapidly determines the extent of infection and points out the dangerous, tolerant carrier as well as the actively diseased animal.

The test is also of great value in dealing with importations of animals for breeding purposes when such animals originate in a dourine-infected area or country.

In countries where the total suppression of the disease is aimed at, every animal declared to be infected, whether on a serum test or by a symptomatic diagnosis, should be destroyed. In countries where the disease is indigenous, the application of the serum test and the permanent branding of every animal infected—if slaughter is not practicable or advisable—would do much to aid the control measures.

E. A. W.

LEISHMANIASIS

The genus *Leishmania* includes protozoan parasites of man and the lower animals, showing considerable resemblance, especially during certain stages in their development, to representatives of the genus *Trypanosoma*. The diseases caused by these parasites assumed far greater importance in the human subject than the case of the lower animals, among which the dog is the only animal that has hitherto been recognized to harbour the natural infection. In human beings *Leishmania* are responsible for the conditions known as Indian kala-azar (or visceral leishmaniasis), which is caused by the species *L. donovani*. Infantile kala-azar, a disease occurring among children to some considerable extent along the Mediterranean basin, was formerly considered to be a morbid entity and attributed to a distinct species, *L. infantum*, but this condition is now identified with Indian kala-azar. Human cutaneous leishmaniasis, oriental boil, or Aleppo button is caused by a separate species, *L. tropica*. Recently a condition which is probably only a variety of ordinary cutaneous leishmaniasis has been found affecting the skin and mucous membranes of human beings in South America. The leishmaniases are not new diseases, Aleppo button was known towards the middle of the eighteenth century, while kala-azar, which was described for the first time in 1882, had probably existed for a long period in the mountains of Garo in India. But although these human diseases had been recognized clinically, their etiology was not discovered until 1903, when it was shown (by Leishman and Donovan) that kala-azar and oriental boil were set up by micro-organisms closely related to one another and belonging to a new genus of protozoa. Investigations carried out since that date have shown that the leishmaniases are not rare diseases confined to a small number of districts in the tropics, but on the contrary have a pretty widespread distribution over the surface of the globe. Inasmuch as the diseases run a chronic course it is also possible for the infection to be carried considerable distances away from the original centres of infection.

Canine Leishmaniasis.—The leishmaniases are manifested in the dog, as in the case of man, under two different forms, viz. the visceral form and the cutaneous form.

Canine visceral leishmaniasis is by far the more common form. The principal and often the only symptom evidenced is wasting. In some animals one may observe towards the end of the disease a paresis of the hind-quarters, the spleen is almost always increased in size, and sometimes is very greatly enlarged. The disease terminates sometimes in recovery, sometimes in death, preceded by sub-normal temperature. The organisms which are the cause of the disease are found especially in the bone marrow and in smaller numbers in the spleen and in the liver. The natural disease is encountered in all those districts in the Mediterranean basin where kala-azar is endemic. Moreover, the *Leishmaniæ* of the canine infection are indistinguishable morphologically from *L. donovani*. It is, however, highly probable that visceral leishmaniasis of the dog and human kala-azar are identical diseases. But the relative incidence of the canine disease and the human disease is inconstant; for leishmaniasis in the dog has been found to occur quite commonly in districts where kala-azar does not exist and, on the other hand, it has not been found at all in India—the country in which human kala-azar is most frequently found. It has also been stated that dogs infected with leishmaniasis are seldom observed in the habitations of human subjects affected with kala-azar.

Cutaneous leishmaniasis of the dog is encountered as a natural infection in certain parts of the world. It is particularly common in Teheran and has been described in Aleppo, Turkestan, and the Caucasus (Laveran). The descriptions given of this condition are very incomplete.

Experimentally, the dog, the Macaque monkey, the white mouse, and some other small rodents are susceptible to infection with the virus of kala-azar and oriental boil.

In smear preparations from the affected organs the parasites are easily recognized in Romanowsky-stained preparations. They appear as small rounded or oval bodies 2-4 μ long by 1-1·5 μ broad, containing two nuclei, the larger of which stains red, while the smaller, which is rod-shaped, assumes a dark violet tint. For the most part the parasites are found intra-cellular, but in smears a few may be also found free-lying. The cells invaded are principally endothelial cells, but they may be also encountered within the polynuclear leucocytes, eosinophiles, and myelocytes. The parasites can be cultivated in the medium referred to above for the cultivation of trypanosomes, viz. in Novy-McNeal medium kept at about 22° C., and a visible growth can be obtained in from two to three days. In this medium, however, the parasites undergo striking changes in morphology and the rod-shaped nucleus grows out to form a flagellum, while the cell body becomes elongated until each organism attains a length of from 15 to 20 μ and a breadth of from 2 to 4 μ. In this stage the parasite thus closely resembles the cultural forms of trypanosomes and other flagellates, viz. Crithidia, Leptomonas, and Herpetomonas. The resemblance between them and the last two mentioned forms, which occur frequently as saprophytes in the digestive tract of insects—lice, fleas, etc.,—is most striking. According to Fantham and Porter the different species of *Leishmania* are probably insect flagellates which have become adapted to intracellular life in man and other vertebrate hosts, and are capable of reverting to the Herpetomonas form in certain conditions, for example, in culture media; but in view of the fact that the *Leishmania* are ordinarily intra-cellular parasites, while the Herpetomonas accomplish the whole of their development in the digestive tract of insects, Laveran maintains that the two genera should not as yet be amalgamated. J. T. E.

FIG. 47.—*Leishmania* (oriental boil). (After Martin Mayer.)

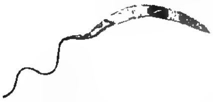


FIG. 48. — *Leishmania* (cultural form). (After Martin Mayer.)

CLASS SPOROZOA

These are exclusively parasitic protozoa which have no cilia or flagella in their growing or vegetative stages, but flagella occur on the adult male element. They ingest liquid food by osmosis, the surface of the body is covered with an ectoplasmic layer or cuticle. In some stages they may execute amœboid movements by throwing out pseudopodia. They may contain one or numerous nuclei, but they have no contractile vacuole as in the case of the Rhizopoda. Propagation by means of spores which in most cases contain sporocysts is characteristic. This Class is divided into two Sub-classes, viz. (1) Telosporidia, and (2) Neosporidia.

Sub-class (1) Telosporidia.—Usually of constant form, rarely amœboid; uninucleate in the mature state; live within host cells in the first stage; spore formation occurs at the end of the life-cycle.

Order (1) Gregarinida.—A body of constant usually elongated form surrounded by a cuticle. In the early stage they lead an intra-cellular existence; in the mature stage they live within the intestine or body cavity of invertebrate animals, especially the arthropoda, and are provided with organelles for adhering to the intestinal wall. Fertilization is isogamous, the spores have coats (chlamydospores) and usually contain several small organisms (sporozoites).

Order (2) Coccidiidea.—Body of uniform spherical or oval shape, lead an intra-cellular life, but are not freely motile in the body cavities. Fertilization is anisogamous. The spores have coats or shells (sporocysts), and usually contain several sporozoites; exhibit a typical alternation of generations; infection of the host takes place *per os* by means of the oöcysts.

Order (3) Hæmosporidia.—Parasites of the red blood corpuscles of vertebrate animals. They also exhibit a typical alternation of generations (schizogony, sporogony), but this is accompanied by a change in hosts, that is, schizogony takes place in the blood circulation of the vertebrate host, while fertilization and sporogony take place in an insect transmitter; the spores are naked. Fertilization is anisogamous.

Sub-class (2) Neosporidia.—These are sporozoa somewhat resembling the Rhizopoda, and in the adult stage are often multinucleate. The form of the body varies exceedingly and is often amœboid, spore formation commences before the completion of growth. This Sub-class is divided into two Orders—(1) the Cnidosporidia, and (2) Sarcosporidia. The Cnidosporidia are again divided into three Sub-orders — Myxosporidia, Microsporidia, and Actinomyxidia. These are parasites respectively of fishes, insects, and marine invertebrates. The Sarcosporidia are elongated parasites of the muscular fibres of mammals and rarely of reptiles. In this situation they form cysts—the so-called Miescher's tubes—very commonly found in the muscle fibres of the domesticated animals, but apparently cause no ill effects; life-cycle still somewhat obscure.

COCCIDIOSES

Diseases caused by protozoan parasites of the Order Coccidiidea.

Coccidia were first observed by Hake (1839) during his investigations on the so-called coccidial nodules of rabbits. Remak (1845) and Lieberkühn (1854) drew attention to their similarity with organisms known as psorosperms found in fishes, and hence the disease caused by them came to be known as psorospermosis. Leuckart (1879), however, showed that parasites of the type found in the rabbit possessed certain well-defined characteristics and named them coccidia, constituting a group of the Sporozoa apart from the Psorospermia (Myxosporidia). In the course of time parasites of this type were found not only in very many vertebrate animals, but also in invertebrates, and a large number of genera and species have been described inhabiting principally the epithelium of the intestine and its appendages and, in much rarer and less important instances, other organs, such as the kidneys, spleen, ovaries, vas deferens, and testicles.

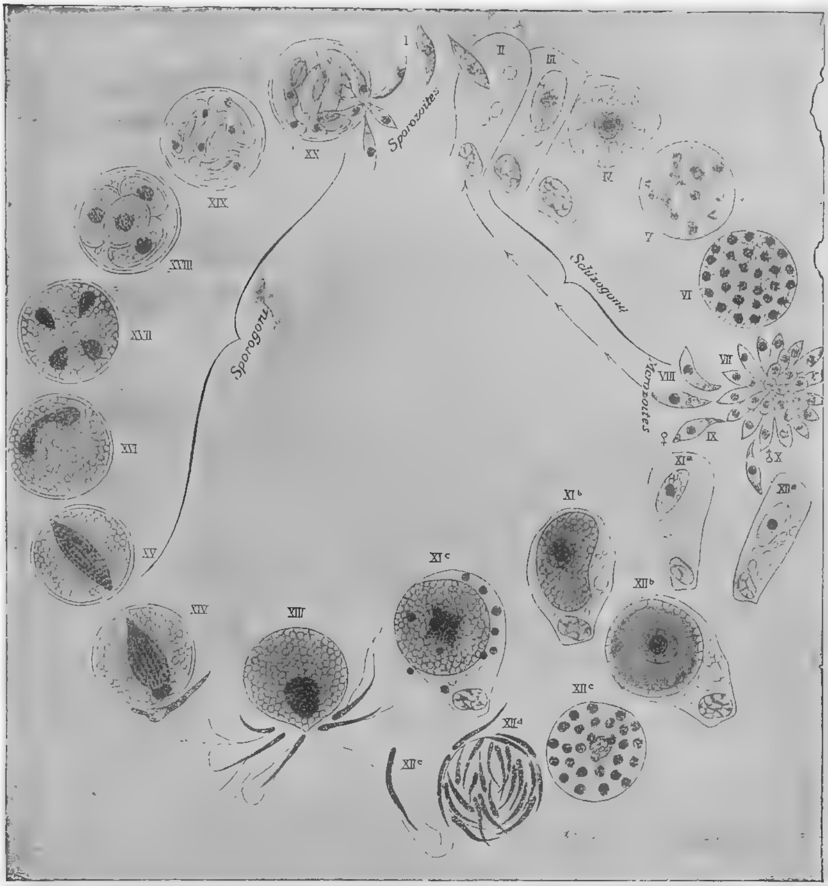


FIG. 49.—Life-cycle of *Eimeria* (*Coccidium*) *schubergi*, Schaud., from the intestine of *Lithobius*. (From Fantham, *Animal Parasites of Man*. After Schaudinn.) The infection is caused by a cyst (XX.), containing spores, which reaches the intestine of a *Lithobius*, where it discharges the sporozoites (I.); II., a sporozoite invading an intestinal epithelial cell; III., intestinal epithelial cell with young trophozoite; IV., intestinal epithelial cell with a globular schizont; V., nuclear segmentation within the schizont; VI., the daughter nuclei arranging themselves superficially; VII., formation of merozoites; VIII., merozoites that have become free, and which, penetrating into other epithelial cells of the same intestine, repeat the schizogony (II.-VIII.); IX. and X., merozoites which, likewise invading the epithelial cells of the same intestine, become sexually differentiated; XI.^{a}, young macrogametocyte; XI.^{b}, older macrogametocyte; XI.^{c}, mature macrogametocyte (discharging particles of chromatin); XII.^{a}, young microgametocyte; XII.^{b}, older microgametocyte; XII.^{c}, increase of nuclei in the microgametocyte; XII.^{d}, the globular residual body around which numerous microgametes have formed; XII.^{e}, an isolated microgamete; XIII., the mature macrogamete surrounded by numerous microgametes and forming a cone of reception or fertilization prominence; XIV., shows the nucleus of a microgamete that has penetrated and fused with the nucleus of the macrogamete (fertilization)—the latter forms a membrane and becomes an oöcyst; XV., XVI., XVII., nuclear segmentation in the oöcyst; XVIII., oöcyst with four sporoblasts; XIX., the sporoblasts transformed into spores, each containing two sporozoites; XX., the cyst introduced into the intestine and liberating the sporozoites by bursting.

The life-history of the coccidia and their mode of propagation within an infected host were made the object of study by a number of investigators, and many points remained in doubt until finally the main facts were elucidated by the classical researches of Schaudinn (1900) on the life-cycle of the millipede (*Lithobius forticatus*), and also to some extent by the work of Léger (1897) on the coccidium of another millipede (*Scolopendra cingulata*), and of Simond (1897) on the coccidium of the rabbit. The complete life-histories of the various coccidia infecting the domesticated animals have in some cases not yet been fully worked out, but in the case of the more important parasites the essential characteristics have been found to be strictly analogous to those of Schaudinn's coccidium of the millipede; the life-cycle of this coccidium is depicted in Fig. 49.

In the course of its life-cycle a typical coccidium thus exhibits an alternation of generations with the production, first, of an indefinite number of elements by an asexual method of multiplication (schizogony) within the epithelial cells of the host, then the elements of division become differentiated into male and female elements (gametes), copulation takes place, and, again, a second method of division (sporogony) takes place when the fertilized female (oöcyst), having gained access to the outside world, becomes divided after a certain interval into a certain number of daughter elements (spores and sporozoites) enclosed in a resistant capsule, forming the egg body or ripe oöcyst. It is this ripe oöcyst that when ingested by a healthy susceptible animal gives rise again, after liberation of its sporozoites, by a process of asexual multiplication within the epithelial cells of the host, to an enormous number of parasites (merozoites), and thus disease is produced, and the cycle eventually repeated.

The more important coccidia of the domesticated animals belong to the genus *Eimeria* (or group Eimeridæ)—that is, to the same genus as the above parasite of the millipede—and this genus is characterized by the facts that the male elements or whip-like processes and the oöcyst divide into four spores, each containing two sporozoites.

Coccidiosis, or the disease set up by coccidia, assumes greatest importance in the case of the rabbit; in this animal the disease is of almost universal occurrence, and causes in certain circumstances serious losses. Of late years the disease has been shown to be of very widespread occurrence among cattle, and may cause fairly serious losses among younger animals especially in some districts. Coccidiosis has also been reported to have occasioned losses among sheep and goats. The disease has long been known to cause serious outbreaks in poultry yards, and in addition it has proved a serious menace in some instances to the rearing of game birds, such as pheasants and grouse, under artificial conditions. Coccidia, belonging to another genus, have been encountered in innumerable wild birds, but it is not known whether they are really the cause of any serious disturbance to the birds. Coccidia of the genus *Diplospora* have been identified in certain localities in the dog, cat, and polecat, but the effects produced appear to be very slight.

Coccidiosis of the Rabbit.—This is usually an enzoötic disease affecting chiefly young rabbits, with symptoms of diarrhœa and wasting; the causal agent is the coccidium *Eimeria stiedæ*, which multiplies in the epithelial cells of the mucous membrane of the intestine and also in many cases in the epithelial lining of the bile-duct and its branches, setting up characteristic lesions in the liver. Although acute symptoms of the disease are only seen in young rabbits, the infection is so widely distributed among domesticated rabbits that it is scarcely possible to obtain a rabbit which does not harbour the parasite. It appears that adult rabbits, either on account of increased resistance or immunity acquired while young, are able to withstand the presence of considerable numbers of coccidia without showing manifest symptoms; but such rabbits are a source of danger, inasmuch as they act as carriers of the parasites and are thus able to communicate the disease in an acute form to their young. Serious outbreaks also frequently occur among wild rabbits and hares; the number of rabbits affected in this country has been estimated at 92 per cent. There is reason to believe that the rabbits of no country or district are exempt from the infection. Generally speaking, however, it may be stated that coccidiosis is of particular importance where the rabbit population is dense and confined in a limited space. The disease thus constitutes a fatal bar to the intensive cultivation of rabbits on a large scale.

Etiology.—The parasite designated *Eimeria stiedæ* has passed under various other names—*Psorospermium cuniculi*, *Coccidium oviforme*, and *C. perforans*. The last two names were given to it by Leuckart, *C. oviforme* being regarded as the form invading the liver and *C. perforans* the intestine; these two forms are now, however, generally held to be identical.

The life-cycle of the rabbit coccidium, so far as it has been made out, corresponds closely with Schaudinn's classical description of the life-cycle of the millipede coccidium, and is briefly as follows:

If one examines a thin film of fæces from an infected rabbit under a low power of the microscope one can easily recognize the adult parasites in the form of small ovoid bodies like immature birds' eggs. Sometimes they are rounded, and they vary in size from about 22-50 μ long by

13-28 μ broad. Each parasite is surrounded by a distinct double contoured envelope except at one pole which is narrower than the other and somewhat flattened. At this point there appears to be an orifice in the capsule (micropyle), but one can sometimes see around this point a mass of mucinous material which acts as a sort of plug. In a small minority of the parasites the protoplasm completely fills the capsule and is coarsely granular, these representing unfertilized parasites. However, in the majority the protoplasm is seen to have undergone considerable shrinking and takes the form of a coarsely granular-looking ball-like mass in the centre of the capsule, and in it a nucleus can sometimes be discerned with difficulty. The remaining space is occupied by a transparent liquid. The entire oöcyst has a slightly yellow tinge, while the ball-like mass may appear slightly greenish or greyish.

The further development of the oöcysts can only take place when they find themselves in suitable conditions as regards moisture and have free access to oxygen; the body temperature also appears to be rather too high. If one thus spreads the fæces out into a thin layer with water, or preferably a 5 per cent solution of potassium bichromate so as to inhibit the multiplication of other organisms, one can observe the so-called sporulation of the oöcysts. The first indication of this process may be seen as early as the second day, and it may be complete on the fourth day. Exceptionally, under favourable conditions, it may be complete within three days. On the surface of the ball-like protoplasmic mass four nearly clear-looking hump-like projections appear, which increase in size at the expense of the remaining protoplasm until this becomes completely divided up into four rounded masses (sporoblasts) and a small residual body (often difficult to recognize). Each of these bodies becomes oval and then pear-shaped and surrounded by a very resistant membrane, and at the narrow end there appears a highly refractile small body (Schneider's corpuscle) whose function possibly is to seal up a potential micropyle at this point. When it has thus developed a resistant membrane the sporoblast is termed a spore or sporocyst, for meanwhile within it are formed two comma-like bodies (sporozoites) and a residual body. The oöcysts are now "ripe," and it is not until they have completed this ripening process that they are capable of infecting another rabbit.

When the ripe oöcyst is ingested by a rabbit it apparently passes unaltered through the stomach, and (according to the researches of Metzner) the sporozoites become liberated first from the sporocysts and then from the oöcysts by passing from the micropyles in each case under the action of the pancreatic juice in the small intestine. The trypsin digests away the thin membrane at the micropyle, and by virtue of creeping movements the sporozoites wriggle out and penetrate by their narrow ends the epithelial cells of the small intestine or of the bile-duct and come to rest in the protoplasm of the cell between the nucleus and the free border. Here each parasite grows in size and becomes rounded in shape. At the same time changes take place within its nucleus, in which the chromatin becomes collected into a vesicular mass and then undergoes a process of division into from eight to fifteen or twenty daughter nuclei distributed in one plane through the middle of the parasite; the protoplasm becomes divided into elongated strips, each enclosing one of these daughter nuclei, so that the parasite (schizont) now bears some resemblance to a peeled orange; each strip (merozoite) then becomes liberated and repeats the process (schizogony) of invading an epithelial cell, growing and dividing into a number of daughter parasites (merozoites) in the same manner as the original sporozoite (recently Reich has figured a distinct fibrillar process at one end of the merozoite and originating from the nucleus by means of which it is able to penetrate the epithelial cells). This process of schizogony goes on an unknown number of times with the resulting invasion of a very considerable number of epithelial cells, but sooner or later it comes to a stop and the merozoites develop into sexual (male and female) elements.

The female element (macrogamete) is recognizable by the fact that its nucleus remains central and undivided while its protoplasm becomes filled with coarse granules, some of which are deeply stained in the same manner as the nucleus itself. The male element can be recognized on account of its large size (it often completely fills the epithelial cell), and its nucleus becomes divided into a very large number (100 or more) of elongated fragments which travel to the surface and develop each at one end a pair of flagella. Each of these bodies becomes liberated and is the male fertilizing element (microgamete). Fertilization takes place by the penetration of one microgamete into a macrogamete; fusion of the nuclei takes place; the macrogamete then becomes surrounded by a slimy substance, and a protective membrane is developed. The fertilized macrogamete thus becomes an oöcyst, which is then passed out in the fæces, and after it has undergone in the outside world the above-described process of sporulation it is capable of infecting further susceptible rabbits when these ingest it with their food. The life-cycle is thus complete.

Symptoms.—These may be acute or chronic and are seen most distinctly in young rabbits. Sometimes the invasion in very young rabbits may be so intense as to produce death apparently quite suddenly, before even oöcysts are present in the fæces. As a rule, however, the disease is manifested by loss of appetite, considerable wasting, rough appearance of the coat, and the animals become dull in appearance. The mucous membranes soon become pale and sometimes assume a jaundiced tint. The belly becomes large, drooping, distended, and at this stage tympanites, dropsy, and especially diarrhœa are the most marked symptoms. The persistent diarrhœa causes more or less matting of the hair beneath and around the anus with crusts of partially dried-up fæces. The rabbit first has an unsteady gait, and later weakness in the limbs becomes more and more marked, until it becomes affected with paralysis of the hind-quarters and finally lies prostrated in an emaciated condition and dies in convulsions. These symptoms may run their course within a very variable length of time, extending from two or three days up to two or three months. As a rule, the older the rabbit the longer does the picture last, while, as stated above, adult rabbits seldom show manifest symptoms of the disease although they may harbour the coccidia and thus act as carriers. On the Continent symptoms affecting the nostrils, pharynx, and middle ear, due to invasion by the parasites, have been described. In such instances the rabbits become affected with a violent nasal catarrh, coughing, and sneezing, sometimes also with angina and otitis ; in the last case symptoms of more or less marked nervous derangement are also seen.

Lesions.—On opening the abdominal cavity the appearance of the liver immediately attracts attention. In advanced cases it is distinctly enlarged; it may attain one-sixth of the total body weight instead of one-twentieth as it does normally. On its surface one can see a number of white patches or spots, many of which are elongated in shape, and they represent bile-ducts greatly distended with coccidia. If one of these spots is pricked with a needle a small bead of milky fluid escapes which, if examined under the microscope, is seen to contain enormous numbers of oöcysts together with epithelial debris. The spots vary in size from that of a millet seed to that of a hazel nut, and they vary considerably in number. When examined histologically one finds in some cases that the irritation and pressure caused by the parasites have produced simply a multiplication of the epithelial cells lining the bile-duct, while in other cases the sub-epithelial tissue is also involved, and delicate, often branching, strands of connective tissue covered with epithelium are seen to grow out from the wall of the cavity, and these may extend right across it from side to side. The epithelial covering is everywhere invaded by coccidia. In some cases the existing epithelium becomes actually destroyed, with the result that the walls of the cavity are devoid of epithelium. The lesions are often confined to the liver and no visible change can be seen in the small intestine. In many cases, however, the mucous membrane of the small intestine shows marked congestion, and in some instances lesions consisting in whitish points or spots of variable dimensions have been described, these spots being formed by masses of coccidia situated in the epithelial cells lining the villi and the crypts of Lieberkühn. Ulceration of the membrane has also been noted. In many instances, especially in rabbits acting as so-called carriers of the parasite, no lesions are found in the liver, although the parasites can be found without great difficulty in the intestinal contents. One may also note in certain outbreaks of the disease a grave intestinal infestation without simultaneous invasion of the liver. It is doubtlessly this point which has led some authors to differentiate between hepatic coccidiosis and intestinal coccidiosis of the rabbit and attribute the conditions to different species or varieties of parasites.

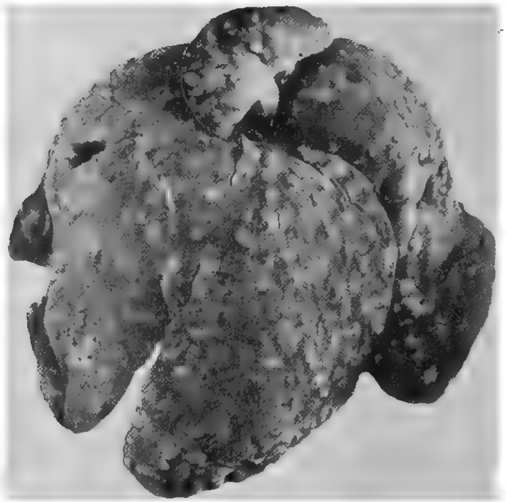


Fig. 50.—Liver of rabbit with coccidiosis.

Diagnosis.—As a rule it is quite easy to make a certain diagnosis during life by examining a thin moist film of fæces under the microscope and detecting the presence of the oöcysts in the form of small ovoid bodies with a highly refractile double contoured envelope. As mentioned above, however, very young rabbits may succumb to the infection before the presence of oöcysts can be detected in their fæces.

Treatment.—In the scene of an outbreak this resolves itself into preventive treatment. It is useless to waste time over attempts at curative treatment, inasmuch as there is no evidence that drugs have any appreciable action in checking the course of the disease, and in the meantime the affected animals, especially those severely affected, are voiding large quantities of oöcysts in their fæces which, after undergoing a process of ripening—a process which may be completed in as short a period as three days—are ready to infect other rabbits and produce the disease. It is thus much better to kill off rabbits showing distinct symptoms so as to diminish the risk of spread. Astringent substances such as tincture of catechu and sulphates of copper and iron mixed with the food have been recommended, but it is difficult to see what effect they could have, particularly in the case of the parasites multiplying high up in the bile-ducts. A rational method of treatment can be easily devised based upon the facts known regarding the life-history of the parasite. One has to bear in mind that infection can only take place by the ingestion of food contaminated with the fæces of an infected rabbit at least about three days previously; the essential conditions for the ripening of the oöcysts are a sufficient supply of moisture and oxygen, adult rabbits may act as carriers without showing manifest symptoms, therefore the obvious method of preventing further infestation of the still healthy-looking rabbits is to separate or destroy the affected subjects and remove them into a cleansed and disinfected hutch every three days. This can easily be accomplished by taking the rabbits out of the contaminated hutch and placing them in another that has been thoroughly cleansed by the use of boiling water or some disinfectant solution such as 5 per cent carbolic acid or 2 or 3 per cent lysol or creolin, or sprinkled over with quicklime after washing; the hutch is then thoroughly dried and its floor sprinkled with fresh clean litter. The rabbits are then introduced into this hutch and in the meantime the first hutch is subjected to the same process of cleansing and disinfection. The rabbits are moved back into it after a sojourn of three days in the second hutch. This process is repeated until the rabbits attain such an age when they are no longer susceptible to the ill-effects of the invasion by the parasites. While the method is being undertaken those subjects which chance to show distinct symptoms are eliminated. Care should be taken not to place young rabbits in the same hutches as older ones which are very likely to be carriers of the coccidia. The same remarks apply to rabbits kept in the open on a soil floor. Change of enclosure should be practised as before, the ground should be freed of parasites by turning its surface and the free use of quicklime or other disinfectant, after having removed the excrement, and care taken to maintain the soil in a porous dry condition, and free from the moisture necessary for the development of the parasites.

Coccidiosis of Cattle (Rote Ruhr, Dysenteria, *Coccidiosa bovum*).—This is a disease usually enzoötic in character and caused by a coccidium, *Eimeria zürni* (*Cytospermium zürni*, *Coccidium zürni*, *Coccidium bovis*). There has been some discussion as to whether this coccidium was identical with that of the rabbit, but recent researches have established its specificity beyond doubt.

The coccidium is remarkable on account of the great variability in its form and the dimensions. Most (Swiss) authors have recognized a rounded form and an ovoid form; the latter is more rarely present and has been regarded as a more advanced form of development. According to Züblin the round forms measure 12-15 μ in diameter and the ovoid forms 20-25 μ by 10-12 μ, exceptionally 30-35 μ by 20 μ. As in the case of the rabbit coccidium the oöcyst has a double contoured capsule 1 μ in thickness, and if it has been fertilized its protoplasm is collected in the form of a ball in its centre. A considerable number of these may be passed in the form of unfertilized macrogametes. Other authors (Degoix, Montgomery, Storch) seem to have noted for the most part ovoid forms. Theobald Smith and Graybill (1918) describe the occurrence of two distinct species in the same subjects, viz. (1) a small form in which the oöcysts are usually ellipsoid, but sometimes ovoid and measuring on an average 18·6 μ by 14·8 μ, which form spores without a residual body, and (2) a larger form with ovoid oöcysts on an average 18·6 μ by 14·8 μ, which form spores without a residual body, but such a body is formed in sporozoite formation. Craig (1919) in Ireland describes egg-shaped large forms, 32 μ by 24 μ, together with a much more considerable number of circular or nearly circular forms, 16-18 μ by 14-16 μ; a small proportion were ovoid, 12-14 μ by 12 μ, but these were merely macrogametes inasmuch as granular contents completely filled the capsule. Although the life-cycle of this parasite within its host has not been much worked upon, there is reason to believe that it is very similar to that of the rabbit coccidium. Sporogony takes place also in much the same manner as in the case of the rabbit parasite; the oöcysts are capable of development even after having remained for more than three months in putrid fæcal material; sporulation requires the presence of air and light. When spread out in a very thin moist layer most of the parasites accomplish their development in three to five days, with the formation of four sporoblasts (+a residual body) which become transformed in sporocysts, each of which afterwards contains two sporozoites (+a residual body). Infection has been set up experimentally in young animals by the ingestion of ripe oöcysts (Guillebeau, Hess); three weeks later slight diarrhœa was noticed, and a diarrhœa lasting from one to three days with the appearance of small numbers of coccidia in the fæces. Montgomery (1912) infected a two-months-old calf with ripe oöcysts, and eighteen days later it showed the first symptoms of coccidiosis, and on the six following days the parasites could be detected in the fæces. Two months later fæces from the first calf containing sporulating oöcysts were administered to two other calves, but without result. Experiments in the transmission of the disease to goats, sheep, rabbits, and guinea-pigs, mice, and fowls have given negative results.

Distribution.—Bovine coccidiosis appears to be very widespread in Switzerland and affects particularly young animals living on the hills and mountains, especially in the Northern Cantons, while it seems to be unknown in the low-lying parts. The disease has also been recognized in certain districts in Germany (where it was first discovered, Pröger, 1878), France, Belgium, Italy, Russia, British Isles (Cornwall, Sussex, Somerset, Scotland, Ireland, and probably many other parts), Africa (Tunis, British and " German " East Africa, Transvaal, Cape Province), Philippine Islands (both in cattle and carabaos), Australia (New South Wales), America (Maine, North-Western States, and New Jersey, U.S.A.), and Panama Canal Zone.

Transmission of Infection.—Infection takes place as in other coccidioses by the ingestion of food material contaminated with the fæces of infected animals, particularly at pasture, and experience shows that damp marshy pastures are especially dangerous in this connection. But in Switzerland, on the other hand, Züblin noted that the disease occurred far more frequently in locations situated at an altitude of from 22,000 to 28,000 feet than on farms in the valleys. In any case the disease occurs principally at pasture, and probably no more than 5 per cent of cases appear in animals kept indoors (Railliet), and it thus in many parts exhibits a marked seasonal incidence; cases commence to appear in June or July twenty to thirty days after the animals are put out to grass, continue throughout the summer, and disappear towards November. Isolated cases may be seen at other times in the year. The disease is seen more commonly during damp years. As a rule the disease is manifested in animals from six months to two years old, and is particularly severe when they are from ten to eighteen months of age (Degoix). It is usually of very rare occurrence in animals of other ages. Recently, however, Smith and Graybill noted the development of the disease exclusively among very young animals; the first symptoms were noted when they were from four to seven weeks old, indicating that infection must have taken place very soon after birth, probably by sucking the contaminated teats of the dams; one case only was found in a calf eleven months old. Ott (1913) noted an outbreak affecting 10 per cent of the young cattle of a herd at pasture, and subjects less than a year old appeared to be immune. Gair (1898) in Scotland observed his cases in animals from one to five years old. According to Hess the disease displays an enzoötic character in young animals, whilst it is of sporadic nature in adults. Otherwise there appears to be no special predisposition as regards condition or breed of the host. Numerous observers, however, have noted that a considerable proportion of animals in an affected herd exhibit what appears to be a natural immunity against the disease. It has also been proved that cattle may have an acquired immunity.

Symptoms.—There are no preliminary symptoms such as fever or loss of appetite, but the disease as a rule commences with signs of diarrhœa. For a day or two liquid, dark green coloured fœtid fæces are evacuated involuntarily. In other cases, however, the first symptom of the disease may be the expulsion of blood-clots among the normal-looking fæces (Züblin). These clots vary in size from that of a pea to a walnut, and during their evacuation the animals may strain and show signs of pain and then resume their normal posture. In milch cows diminution of the milk yield is seen from the commencement. In a day or two when diarrhœa exists the fæces become mucous in character; blood is expelled in greater quantity and the clots attain the size of a hen's egg, and a mass of clot may be passed at the end of the act of defæcation, which is more frequent and straining more marked. There is scarcely any fever, and the pulse is slightly accelerated. The symptoms rapidly become worse and the animals become dull and exhibit loss of appetite and great thirst, rumination becomes irregular, and champing of the jaws is often noted. The blood-clots then increase up to the size of a fist, the fæces become very fluid and offensive. The temperature rises just a little (about 102° F.); pulse 70-80, respiration 20-25 (Railliet). There is rapid wasting, the coat becomes staring, there is weakness of the hind-quarters, the animal has difficulty in keeping on its feet, and in getting up. This acute stage may last from five to ten days, after which the disease progresses towards recovery or death. Recovery is more common in older animals than in young, and the rapidity with which it is produced varies inversely with the length of the period of acute symptoms. Usually the expulsion of blood stops on the sixth or the eighth day; straining may still last for a few days and diarrhœa may continue for two or three days, or even for two or three weeks. The appetite may remain capricious for a long time and the general condition may vary from time to time. In other animals, especially younger animals, the symptoms are aggravated on the other hand with great rapidity. The rectum, which is often prolapsed, appears red and swollen, the fæces tend to resemble urine and contain besides blood shreds of mucus. However, the blood disappears almost as quickly as in favourable cases, but it may be replaced for some time by fibrinous material. The appetite and rumination cease completely, the animals stand with outstretched neck, raised tail, and hollow flanks, sunken eyes, and often swelling of the eyelids. Temperature usually does not rise above 102° F., but it may exceptionally rise to 2° or 3° higher, but it furnishes no indication as regards the severity of the invasion. Pulse rises from 80 to 120 and even 140 per minute. At this stage the animals may exhibit a somewhat depraved appetite. Finally, they remain lying down in a prostrated condition and die of exhaustion. This description refers to the most frequently observed form of the disease—what may be called the " acute " form; other forms, which have been classed as " hyper-acute " and " chronic," have also been observed, and certain authors (Theiler and Montgomery) have described particular symptoms such as lachrymation, buccal ulceration, and cutaneous eczema which tend to give the disease a very considerable clinical resemblance to rinderpest. The clinical picture may also be masked by the simultaneous appearance of other affections such as bronchitis, pneumonia, eclampsia, paralysis or black-quarter, and in Africa the disease has been frequently noted associated with rinderpest.

The duration of the disease varies considerably, depending upon the degree of intensity of the invasion. Moreover, between the hyper-acute forms and those running a slow course one may observe all kinds of intermediate forms. Although death may occur within twenty-four hours, usually it does not occur until after from one to two weeks, and more rarely after three weeks. In the same manner recovery in mild cases may take place in three or four days, whilst in other cases the convalescent stage may be prolonged for up to five or six months, and at the end of this period the animals may die of secondary anæmia. Montgomery (1913) demonstrated experimentally that one clinical attack produces a powerful immunity against subsequent attack, so that the parasite may remain in a latent stage within the alimentary tract and not give rise to any symptom. However, the onset of piroplasmosis or anaplasmosis was found capable of breaking down this immunity, and afterwards oöcysts could be discovered in abundance in the excreta.

Diagnosis.—In an enzoötic centre the clinical characters of the disease are in the majority of cases sufficient to enable one to establish a diagnosis. However, a certain diagnosis can only be made by microscopic examination of the fæces, and, as a rule, this presents no difficulty; a thin film of fresh fæces is examined under a low power of the microscope and a search is made for coccidia. As a rule these cannot be found in the pure blood-clots, but they are found in abundance in the shreds of mucus covering the fæces (according to Zschokke, often to the extent of 50 to 100 per field). Smith and Graybill have described a method for concentrating the fæces in cases where coccidia are present in very small numbers.

Prognosis.—The chances of recovery are admittedly more restricted in the case of young and weak animals than in older and better conditioned animals. However, prognosis must be guarded in cases where loss of appetite and rumination are manifested early. Sometimes the death-rate is fairly high (Züblin estimates it at about 5 per cent, Hess at from 8 to 13 per cent). The flesh of animals slaughtered in the early stages can be safely utilized for human consumption.

Lesions.—Züblin describes these as follows: In animals killed after the disease has been in progress three to four days the changes are confined to the rectum, the mucous membrane of which is thickened and congested, and the epithelium detached in many points so as to present a wrinkled surface dotted over with hæmorrhagic points and often covered with blood-streaked mucus. The surface of the rectal contents, which are normal in consistence, are covered with small clots of pure blood. In cases killed after five or six days rectal lesions are again the only ones noted in most cases, but sometimes the mucous membrane of the colon and cæcum is congested and thickened. The bowel contents are the consistence of clear soup mixed with shreds or flakes of clotted blood, the spots denuded of epithelium have increased in size. In cases lasting from six to eight days the cæcum and colon are somewhat congested and their contents very liquid. The mucous membrane of the rectum shows only slight hyperæmia and no hæmorrhagic spots, but is hard and coarse-looking where it has been denuded of epithelium. The intestinal contents are greyish-red and mixed with shreds of mucus, but contain no blood-clots. In cases lasting from eight to ten days the intestine is affected throughout, although the rectum still shows the most marked changes. The mesenteric glands are enlarged, the mucous membrane of the large intestine is wrinkled and covered with brownish-grey patches and shallow ulcers where the epithelium has been shed. The contents are liquid, have an offensive odour, and are very small in quantity. There is extreme emaciation and the muscles are macerated in appearance and the heart flabby and empty.

On histological examination of the rectum one notes, in the first stage mentioned above, congestion of the blood-vessels and the presence of small granular bodies, probably coccidia in the glandular cells; the intestinal contents show very few coccidia. In the second stage, there is no longer any congestion of the blood-vessels, but considerable areas are denuded of epithelium and many of the crypts of Lieberkühn are destroyed whilst others are dilated and filled with exudate; the fæces mixed with blood and mucus contains the parasites in large numbers. In the third stage, the cæcum and colon show no change except congestion, but the rectum is found to have completely lost its epithelium; the crypts of Lieberkühn appear as if they had been pressed together and ruptured and show no cellular covering except in their deeper parts. The cells are dilated by the presence of coccidia, but the parasites are found for the most part free-lying within the lumen of the glands; the parasites are very numerous in the intestinal contents. In the fourth stage, the rectum shows absence of epithelium and leucocyte infiltration as before, but the changes are deeper and there are large cavities extending down to the submucous coat. The crypts are still further reduced in size and number, but a few coccidia are still found in the remaining cells. In the other parts of the intestine the lesions are of the same nature but less marked. A few authors have found coccidia in the small intestine.

Treatment.—(*a*) Prophylaxis. The same principles apply as in the case of coccidiosis in the rabbit. In enzoötic centres cattle, especially young cattle, should be removed away from damp pastures and especially those recognized as infective. Affected animals should be completely separated from those still apparently healthy, and on an infected farm the young animals should not be allowed to run with or follow after at pasture adult cattle which may act as "carriers" of the parasite. Railliet insists upon the importance of keeping susceptible animals away from marshy or boggy land and giving them clean, artificially drawn water for drinking purposes. However, inasmuch as the parasite is capable of "ripening" even when placed in the thinnest film of moisture, it is quite as likely that it can find the conditions necessary for development on moderately dry pasture, particularly when one has to consider that such moisture may be afforded during a considerable part of the day by the dews. When the disease occurs among young animals kept loose indoors it is important to keep the floor dry, to remove the bedding frequently, and to disinfect the excreta. Hutyra and Marek recommend the use of 3 per cent sulphuric acid for spraying the fæces; but Galli-Valerio has shown that certain forms remain more or less intact for three days after treatment with 50 per cent of this acid; this author also showed that intense cold did not completely destroy the parasite, and even the deep frosts of the Swiss mountains only had the effect of suspending their development.

(*b*) Curative. Certain forms of specific treatment have been recommended and the following have been applied in practice. Smythe uses the following mixture:

℞ Pulv. catechu, ʒii.
Pulv. asafœtidæ, ʒi.
Tct. opii, ʒii.
Creosoti, ʒi.
Aquam ad, Oss.
M. ft. haust.

To be given in a pint of gruel and repeated twice daily. Smythe carried out the above treatment in conjunction with injections of adrenalin to control the blood-clots.

Some Italian workers (Perroncito, San Lorenzo, Cremona) appear to have obtained very successful results after the use of thymol. The first two authors administered this drug in capsules each containing from seven to eight grammes of the drug. The administration of two capsules a day to calves a few months old is stated to have brought about extremely rapid improvement; in two or three days the coccidia disappeared from the fæces and the more acute symptoms were remarkably diminished in intensity.

The use of drugs to combat the symptoms, especially diarrhœa and wasting, is strongly indicated, and any of the ordinarily employed opiates and vegetable and metallic astringents, mucilaginous emollients, and intestinal disinfectants can be recommended. Hess prescribes the use of enemata consisting of a weak solution of nitrate of silver, sulphate of iron or sulphate of zinc, together with opium. Salvisburg advises the administration of animal charcoal mixed with tannoform and bismuth sub-nitrate. Smythe injects adrenalin subcutaneously to counteract the rectal hæmorrhages; Moussu advises ergotine or a solution of gelatine for this purpose. In chronic forms and in prolonged convalescence endeavours should be made to prevent wasting by the use of tonics and special dieting.

Coccidiosis of Sheep and Goats.—In these animals coccidiosis appears to be somewhat limited in distribution. It has been reported upon in France and Northern Africa, and cases have been reported in both animals in this country.

The coccidium of sheep is called *Eimeria faurei*, and of goats *Eimeria arloingi*. The coccidium of sheep is peculiar in that it forms very large schizonts in the mucous membrane of the small intestine which contain enormous numbers of small merozoites. It has recently, however, been doubted whether such parasites are in reality coccidia at all. Disease affects particularly young animals—lambs and kids—with symptoms resembling those seen in coccidiosis of rabbits and ox. The lesions may take the form of innumerable white specks or spots in the mucous membrane of small intestine and sometimes in large intestine. Sometimes the lesions are of a peculiar character and take the form of a number of papilliform outgrowths of the mucous membrane, each about the size of a pea. These outgrowths, or wart-like excrescences, are formed of a connective tissue core covered in the folds of mucous membrane in which numerous coccidia can be recognized.

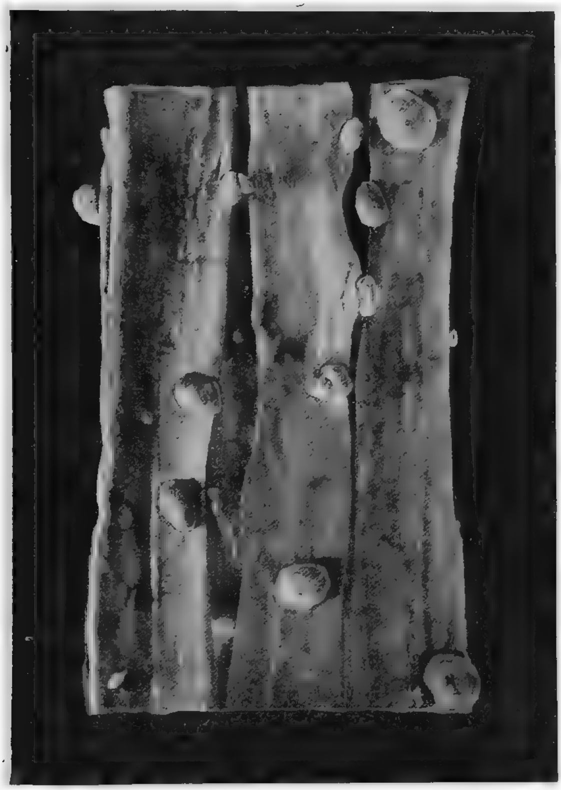


FIG. 51.—Coccidiosis of intestine of goat, showing papilliform growths. (Specimen in the Museum of the Royal Veterinary College, London.)

The mucous membrane elsewhere sometimes shows quite distinct congestion.

Adult sheep and goats have been proved to harbour the coccidia in their fæces without showing any manifest symptoms of disease.

Treatment.—Where local outbreaks have occurred preventive treatment as recommended for rabbit coccidiosis has proved very effectual.

Coccidiosis of the Dog and Cat.—In the dog, cat, and polecat coccidia have also been recorded, but they belong to the genus *Diplospora* or *Isospora*. The species is known as *Diplospora bigeminum*. They differ from *Eimeria* in that two spores or sporocysts only are formed but inside each sporocyst there are formed four sporozoites. In the dog in America these coccidia have been shown to produce only very slight symptoms. J. T. E.

Coccidiosis of Fowls.—This is a severe disease and affects all the domesticated poultry—fowls, ducks, turkeys, geese, pigeons—and also pheasants and grouse reared under artificial conditions. The disease sometimes becomes so severe that 90 per cent of the birds become affected and sometimes all the younger ones die off. It is commonly known as White Diarrhœa of chicks, but it has been found that some bacterial diseases are included under this term.

The parasites affecting all poultry are probably identical, and are termed *Eimeria avium* or *tenellum*. The life-cycle has been pretty fully worked out and corresponds almost exactly with that of the rabbit. The oöcysts are shorter and more rounded in shape than those of the rabbit, measure from 20 μ to 30 μ in length by 12 μ to 20 μ in breadth. The residual body is smaller and the capsule is thinner.

Symptoms.—Diarrhœa and wasting. The lesions to commence with affect the duodenum, and they later spread backwards, and finally involve the cæca severely. In some cases a croupous or diphtheritic inflammation of the cæcum may be observed. The disease may be classified as acute, sub-acute, or chronic. Usually the younger the bird the more acute the symptoms. Adult birds may act as carriers without showing any symptoms.

Treatment.—Chiefly preventive. In a badly affected locality it is best to kill off all fowls and keep free for about a year. Curative treatment in form of administration of astringents such as sulphate of iron in the drinking-water has been recommended.

Coccidia are very common parasites in all kinds of wild birds, but they appear to belong to a different genus and are not known to produce severe symptoms.

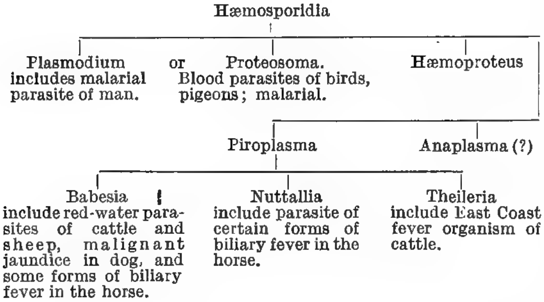
Coccidia have also been reported in other tissues than the intestine: *e.g.* in renal epithelium of goose (*Eimeria truncatum*).

Blackhead in Turkeys.—Not uncommon in this country. Very common in the United States of America, Canada, and South America.

The lesions are found in the liver and cæca. In the former they take the form of white necrotic patches up to the size of hazel nuts or even walnuts. Necrotic patches are also found in mucous membrane of cæca. Symptoms are intense diarrhœa and wasting. Birds become dull and dejected and stand with feathers erect, and a dark or black discoloration of the comb and wattle is observed. The nature of the causal parasite has been the subject of considerable discussion. It was first considered to be an Amœba (*Amœba meleagridis*) (Theobald Smith). Amœbæ or amœba-like structures may be seen in liver and cæca lesions. Later, it was stated that these parasites might be developmental stages in the life-cycle of coccidia, but this has been proved not to be the case. Recent researches have proved it to be a flagellate protozoon which appears to be closely allied to the genus *Trichomonas*, but its exact zoological position has not yet been determined.

Hæmosporidia

The order Hæmosporidia, or parasites of red blood corpuscles, contain a number of genera, of which the following are the more important representatives :


Hæmosporidia
Plasmodium
includes malarial parasite of man.
or
Proteosoma.
Blood parasites of birds, pigeons ; malarial.
Hæmoproteus
Piroplasma
Anaplasma (?)
Babesia
include red-water parasites of cattle and sheep, malignant jaundice in dog, and some forms of biliary fever in the horse.
Nuttallia
include parasite of certain forms of biliary fever in the horse.
Theileria
include East Coast fever organism of cattle.

In Hæmosporidia two hosts are necessary. Schizogony takes place in vertebrate host, and sporogony in the tissues of a biting fly or tick.

Plasmodium and Proteosoma (malaria of man and birds).—In human malaria the biting fly is a mosquito belonging to the genus *Anopheles*. In bird malaria it is a gnat, *Culex*. When these flies bite man or bird respectively they inject into the blood stream a sporozoite which proceeds to enter a red blood corpuscle inside which it loses its spindle shape and becomes rounded or irregular and amœboid, and grows until it fills the greater part of the red corpuscle (trophozoite stage). The growing trophozoite elaborates a large number of melanin granules. The nucleus divides into a number of small pieces and the cytoplasm becomes divided off round each small nucleus. This is a schizont and each piece a merozoite. These merozoites are set free and commence to invade the corpuscles exactly in the same manner as the original sporozoites. This process of schizogony is repeated a number of times, depending on the species of *Plasmodium*, but finally comes to an end, and gametes or male and female elements are formed. The female or macrogamete contains a centrally placed nucleus, whereas the male element or microgametocyte has its chromatin distributed in small fragments round the periphery. No further development takes place inside the vertebrate host. When infected blood is taken by the mosquito or gnat, the microgametocyte undergoes in the stomach of the fly a process of so-called " flagellation." Whip-like bodies corresponding to macrogametes are set free and each proceeds to fertilize a macrogamete. After fertilization certain changes take place in the latter (now known as a zygote), which becomes elongated, oval in shape, and, by virtue of creeping movements, it burrows through the wall of the stomach and establishes itself on the external wall of the stomach. It is now known as an oökinete and it then becomes rounded and rapidly grows in size. Its nucleus becomes divided into a number of fragments and the cytoplasm is cut off to surround each daughter nucleus, with the resulting formation of a number of sporoblasts. The parasite is recognizable to the naked eye as a white body about the size of a pin's head. The nucleus of each sporoblast undergoes further division into a considerable number of daughter nuclei, and around each the protoplasm becomes cut off so as to form a number of spindle-shaped elements or sporozoites. The sporoblast thus becomes a sporocyst or spore. The body then ruptures. The sporocysts are set free in the cœlomic cavity of the fly, and eventually the sporozoites are liberated and they travel forwards and establish themselves in the salivary glands of the fly. There they remain until the mosquito or gnat bites a human being or bird and sporozoites escape with its saliva into the tissues of the host, invade the blood stream, get into the red corpuscles, and the process of schizogony again takes place.

In man there are three types of parasites : (1) *Plasmodium vivax*, which takes forty-eight hours to complete its growing period ; a relapse may thus occur every third day. The disease produced is known as tertian malaria.

(2) *Plasmodium malariæ*, which takes seventy-two hours before schizogony occurs, and a relapse may therefore occur on the fourth day. The patient is said to suffer from quartan malaria.

These two species are the cause of simple or benign malaria.

(3) *Plasmodium falciparum* or *præcox*, the cause of pernicious malaria, in which division takes place only in the capillaries of the internal organs such as the spleen, and fever occurs at intervals which vary considerably. The disease

is termed malignant tertian, irregular, or quotidian fever.

In mild or benign malaria the parasites in the blood take the form of small rounded organisms. The parasite of pernicious malaria can be distinguished because the gametocytes are crescentic in shape.

The parasite of birds is known as *Plasmodium* or *Proteosoma relictum*.

Hæmoproteus.—There is a malaria of birds caused by a parasite known as *Hæmoproteus* or *Halteridium*, the life-cycle of which is of interest inasmuch as it throws light on the development of the parasite of East Coast Fever in cattle. It differs from *Proteosoma* in that the disease cannot be transmitted by direct inoculation of blood. The parasites occur in the blood corpuscles either as rounded parasites or as dumb-bell-shaped structures with a nucleus in the centre. The dumb-bell-shaped structures are gametocytes, and it is difficult to distinguish male from female. The rounded structures are growing forms of the gametocyte. Transmission takes place by a fly of the family Hippoboscidæ. (The particular genus usually incriminated is known as *Lynchia brunea.*) When this fly sucks blood it takes up corpuscles containing the gametocytes, and in the stomach of the fly copulation takes place between the male and female elements, with the production of a zygote. No flagellation appears to take place. Nothing further takes place in the body of the fly. When the infected fly bites another susceptible pigeon the stomach contents are vomited and the fertilized zygote enters the blood stream, penetrates the leucocytes, and gets carried away to the capillaries, of the lung especially. The parasite there increases in size, its nucleus divides, and ultimately a tremendous number of sporozoites is formed within each parasite. These bodies rupture, and male and female elements escape into the blood stream and invade the red corpuscles.

These parasites infect most commonly pigeons, and the particular species in this case is known as *Hæmoproteus columbæ*.

PIROPLASMOSES

Piroplasmosis of Cattle. Red-water or Texas Fever.—The parasite was discovered and described in 1893 by Smith and Kilborne in America. They called it *Pyrosoma bigeminum.* A few years before that (1888) Babes in Roumania had seen small coccus-like points in blood corpuscles of cattle affected with red-water, and he thought they were bacterial in nature; he called them *Hæmatococcus bovis.* Smith and Kilborne demonstrated that these bodies were protozoans in nature and tick-transmitted. The name now generally given is Piroplasma; but again, in recent years it has been shown that all piroplasms are not of the same nature and there has been a tendency to divide them into separate genera. The piroplasm of cattle red-water is therefore now called *Babesia bigeminum.* The Texas fever parasite or the parasite of tropical red-water occurs in the corpuscles in its most typical form as a paired pear-shaped parasite. Each is about 4 μ in length, and it contains a cytoplasm which stains blue with Romanowsky staining and one or two small chromatin masses which stain bright red. Irregular forms often occur in the form of rounded or irregular single parasites; crescentic forms, signet-ring forms, and frequently single pear-shaped organisms are seen within a corpuscle. Until a few years ago the parasites of red-water throughout the world were considered to be most probably identical, but it has been proved since that the commonly occurring parasite of European red-water in particular shows fairly considerable differences (McFadyean and Stockman). In its typical form this parasite also occurs in pairs within a single corpuscle, but the parasites are smaller and considerably narrower and inclined towards each other at a far more obtuse angle. Irregular forms are also met with quite frequently, and it has been proved that immunity set up towards this parasite does not protect against an attack of tropical red-water. This parasite was called *Piroplasma* (or *Babesia*) *divergens* by McFadyean and Stockman, but other authors maintain that the name *Babesia bovis* should be retained for it, inasmuch as this was the specific name originally applied to the European organism before the differences between it and the cause of tropical red-water were pointed out. *Babesia bigeminum* is probably identical with that found in Argentina (*B. argentina*).

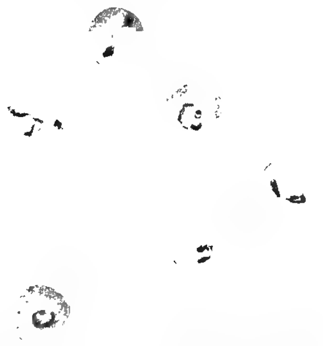


FIG. 52.—*Piroplasma bigeminum* (*? divergens*). (After Schilling.)

Usually in an attack of red-water about 10 per cent of the corpuscles are invaded with piroplasms. Very exceptionally up to 50 per cent have been noted. Often only 1 per cent can be found in a blood examination.

There is no method of cultivating piroplasms, although it has been stated that the piroplasm of the dog has been cultivated by placing infected blood in broth containing a certain proportion of glucose, but probably the authors who described these forms mistook degenerative processes in the corpuscles for the parasites.

In an animal dead of red-water the piroplasms are generally all dead in twenty-four hours, but if blood is taken and preserved in an ice-chest the piroplasms may remain alive for a considerable time—up to fifty days.

The red-water piroplasm is very specific. Only cattle can become infected, but it appears to have no predilection as regards breed or condition. On the other hand, cattle vary greatly in susceptibility according to their age. In calves the piroplasm seldom or never sets up a fatal attack, whereas in adult cattle the piroplasm, especially that of Texas fever or tropical red-water, almost invariably produces a severe reaction. Thus susceptibility increases with age.

Method of Infection.—(1) Experimental. The disease can be transmitted by direct inoculation of blood, and inoculation is capable of setting up a reaction for a long period, up to thirteen years after the recovery of the animal affected. (2) Natural. Exclusively tick-transmitted.

Lesions.—In cases that prove fatal within a week the blood is anæmic and watery; the spleen is constantly enlarged to two or three times its normal size, but never to the same extent as in anthrax. The liver is constantly enlarged, jaundiced, and shows signs of fatty degeneration. There are also petechiæ on the mucous and serous membranes. In cases that last for a longer time spleen lesions are not so marked, and eventually, if an affected animal remains alive for two or three weeks, the spleen may have regained its normal size. Blood changes are those seen in profound anæmia. The corpuscular count may decrease from eight millions to half a million in extreme cases. There is inequality in size and shape of the corpuscles, which may become very granular (punctate degeneration), also the presence of nucleated corpuscles.

Treatment.—Preventive. Cattle can be inoculated against red-water by taking advantage of the fact that young cattle are comparatively immune while susceptibility increases with age. Recovered cattle also harbour the parasites in their blood for a long time afterwards, perhaps throughout life. Before cattle are sent into badly infected centres one thus inoculates them subcutaneously with a small dose of blood from a recovered animal. After inoculation a rise of temperature takes place on the seventh or eighth day and the reaction lasts in some cases for a week. In other cases the reaction is so mild that nothing beyond a slight rise in temperature is noticed. If the symptoms threaten to become too severe one controls the reaction by injections of trypan-blue. This is dissolved in a 1 per cent solution in warm water and about 100 c.c. is injected subcutaneously or intravenously. This dye has been found to be a remarkable specific against the dog piroplasm, and there is a good deal of experience to show that it is also effective against red-water caused by the *P. bigeminum.* Against *P. divergens* it seems to have very little effect.

The immunity thus conferred is not an immunity in the strict sense of the word, for the recovered animals remain carriers of the piroplasm for the remainder of their lives and would be a source of danger if introduced into a clean area. Many countries, such as India, Mesopotamia, and South Africa, are so badly infected with red-water, however, that it is advisable to inoculate all cattle exported from this country to such places.

The eradication of red-water from an infected area amounts to the carrying out of measures for dealing with the transmitting tick. One may attempt either (1) the total eradication of the tick, or (2) the cleansing of the tick of its piroplasm infection.

In America tick eradication has been carried out on a very large scale. Nearly three-quarters of a million square miles in the Southern States had become infested and in 1906 co-operative eradication was commenced. It is contemplated that by 1921, or at the latest in 1923, the whole of this area ought to be free of the tick. The transmitting tick there is *Margaropus* (*Boophilus*) *annulatus*, a continuous feeder which remains attached to its host for a period of about three weeks. In order, therefore, to catch the tick on its host, tick-destroying measures should be applied at intervals of less than three weeks. Dipping for the destruction of ticks is therefore practised every fourteen days during the tick season, from spring to autumn. The dipping preparation used for tick destruction is an arsenical solution, the most effective being a solution of sodium arsenite. This is a constituent of various formulæ recommended, and also of some proprietary preparations such as Cooper's Dip. One formula of a dip used in Queensland (where ticks had become so numerous as to cause great loss in condition of cattle through their bites) is

Arsenic, 8½ lbs.
Caustic soda, 4 lbs.
Stockholm tar, ½ gall.
Water, 400 galls.

The arsenic and soda are boiled in a little water first and then added to the remainder of the water.

The South African formula is

Sodium arsenite, 1 lb.
Soft soap, ¾ lb.
Paraffin, 2 lbs.
Water, 100 galls.

Cattle should be passed through a dipping tank about 35 feet long, containing 2500 to 3000 gallons of dip. In badly infested countries dipping is now very commonly resorted to, not only to eradicate red-water but also for the eradication of the ticks themselves on account of the loss of condition which they cause.

The application of the tick-destroying fluid by means of spraying has been advocated in some places, but this is not so effective as dipping and is not so practicable when large numbers of cattle have to be treated.

In this country these measures could not be adopted in practice for the eradication of red-water, because dipping would have to be carried out at intervals of from three to five days. The transmitting tick, *Ixodes ricinus*, is an interrupted feeder and during some of its development does not remain attached more than three to five days. The problem of tick eradication is therefore very difficult, but measures can be formulated if one bears in mind a few facts in the life-history of the tick. The tick lives almost entirely on uncultivated land, and especially in the neighbourhood of woods and shrubs, and its period of activity is from May to September. Measures should therefore be adopted having for their object the clearing of woods or the avoidance of the known infected parts during the tick season. In some places the freeing of the pastures for a period of at least a year from cattle has been attempted. During this time all the larvæ should die out, but the difficulty is that larval ticks may attach themselves to other animals and thus retain their vitality. In so far as the eradication of red-water is concerned, however, this does not matter much, for there is reason to believe that if the tick is made to bite a non-susceptible animal for a long period of time it cleanses itself of the piroplasm. On some farms housing of cattle for this period of time has been strongly recommended, whilst the infected pastures are grazed over by sheep or horses.

Attacks of red-water are apt to recur if the animal's condition is depressed for some reason. In the tropics acute red-water symptoms may thus be seen in cattle concurrently with those of rinderpest. In this country difficult parturition is apt to bring about the same effect, so that in some districts " parturient red-water " is not uncommon. The causation of this condition, so-called " parturient red-water," however, does not appear to have been definitely connected with piroplasmosis in many cases. J. T. E.

Red-water of Cattle in the British Isles. Bovine Hæmoglobinuria. Black-water. Blood Murrain. Moor Ill. Muir Ill.—Red-water is a disease affecting cattle in which the most prominent symptom is the presence of free hæmoglobin or blood-pigment in the urine and due to a protozoan parasite in the blood called a piroplasm.

Etiology.—The disease in the British Isles is caused by two different piroplasms, viz. the *Piroplasma* (or *Babesia*) *divergens* and the *Piroplasma* (or *Babesia*) *bigeminum*, the former being the more common. As in the case of other piroplasmoses, the parasites occur mainly in the red corpuscles of the blood and are transmitted from animal to animal by means of ticks, principally *Ixodes reduvius* (*I. ricinus*) and *Hæmaphysalis punctata.*

Red-water chiefly attacks adult cattle, feeders and milkers, while calves appear to escape to a great degree. The latter, however, are not immune and probably in infected districts most of them are subjects of very mild attacks which escape observation. Cattle reared in infected localities possess a certain degree of immunity, or rather tolerance, owing to mild infections in early life, but animals introduced into those districts from localities free of the disease are readily infected and suffer to a varying degree. The disease occurs principally on rough uncultivated lands, such as moorlands, districts favourable to the development of intermediary bearers. It is consequently less frequently met with in high, dry, and well-cultivated districts. The disease occurs especially in the warm season from the end of May up to October, although severe attacks may be encountered during the winter.

The period of incubation in natural cases is about a fortnight after the animals have been brought on to affected pastures. This period may be reduced to seven days in cases experimentally produced by the intravenous injection of 5 to 10 c.c. of virulent blood.

The mortality of British red-water varies from 1 to 10 per cent, the average mortality being about 5 per cent. Animals recovering from red-water develop considerable tolerance to subsequent infection, but as they harbour the organism they remain centres of infection. Moreover, an attack of any acute febrile or debilitating affection is likely to cause renewed virulence of the parasites and set up a severe attack of red-water.

Symptoms.—Two types of the disease may be recognized, an acute or malignant type which is frequently fatal and a mild or benign form which is seldom fatal.

The *acute type* is usually sudden in occurrence, the affected animal is very greatly depressed, and isolates itself from the rest of the herd, with nose extended, dribbling, grunting, grinding the teeth, back arched, and moving about, if at all, in a very listless fashion, often with a staggering gait and swaying of the hindquarters. The coat is dry, harsh, and staring, and not infrequently the animal is in a hidebound condition. The respiration is rapid and blowing and may be from 90 to 100 per minute. The heart-beats are very loud and can in many cases be heard beating five or six feet away from the patient. The appetite is lost and rumination ceases. The visible mucous membranes are pallid and may have a yellow tinge. The eyelids, lips, anus, and vulva are drawn and wrinkled. The pulse is jerky, weak, small, and frequent, hovering about 100 per minute. There is almost invariably diarrhœa at the beginning, the fæces being extremely watery and ejected with considerable force. This condition may pass into one of obstinate constipation, and that is usual in those cases that terminate fatally after two or three days. With regard to that point practical farmers will often say that they do not so much mind "Blood Murrain" as long as there is not also "Dry Murrain" (obstinate constipation, generally referred by them to the omasum or manyplies). The external temperature is uneven, some parts of the skin hot and others cold. The internal temperature taken per rectum varies considerably. It is high at the outset of symptoms, and if taken twice daily will usually be found for a few days oscillating, being higher in the evening than morning (103·5° F. to 107° F.). After three or four days the morning temperature will be the same as the evening, and will remain elevated until the disease terminates fatally or in recovery. In the former case the temperature may fall to 2° or 3° below the normal just before death, and in the latter case the restoration to the normal may be very rapid. The urine is in the vast majority of cases, but not constantly, pigmented with hæmoglobin to a varying degree. It may have a bright claret colour, or it may appear almost opaque and black, somewhat like stout. In favourable cases it may clear up almost as suddenly as it became coloured. It is generally albuminous, and consequently forms a froth like soap-suds on falling to the ground, the froth remaining for hours. In milkers lactation diminishes and the milk is not infrequently tinged pink.

On affected animals more or less numerous ticks can generally be found in various stages of development, and varying considerably in size. They may be on the skin at any part, but are more commonly found on the dewlap and inner sides of the fore- and hind-legs, and firmly attached, their respective heads being buried in the skin. Small nodules, somewhat painful to the touch, may also be found, marking previous seats of attachment of ticks.

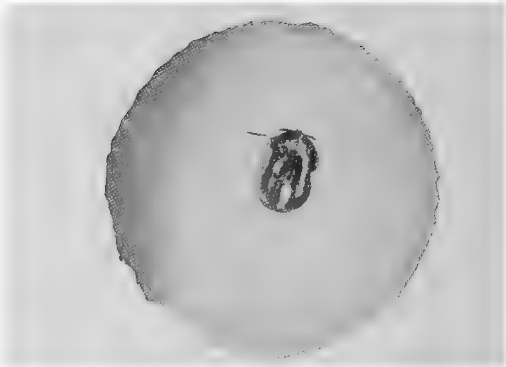


FIG. 53.—*Ixodes ricinus.* Adult female carrying male *in coitu.* (Natural size.)

As the disease advances the blood becomes very thin and watery and on exposure to the air usually darkens quickly. On coagulation the serum is normal if the acute stage has passed, but if the blood be drawn during the acute attack the serum is red owing to the hæmoglobin in solution (hæmoglobinæmia). In defibrinated blood there forms a lower purplish-red layer and an upper lighter red layer, whereas in normal defibrinated blood the upper layer is generally yellow.

On microscopic examination of blood films prepared during the febrile stage and appropriately stained, the parasites can be seen in the red blood corpuscles (see description of "Protozoa"). Another pronounced change is the diminution in the number of red corpuscles, and the loss bears a direct relation to the severity of the attack. The normal number is about eight millions per cubic millimetre. In mild cases the destruction may go on from four to eight days and the number be reduced to

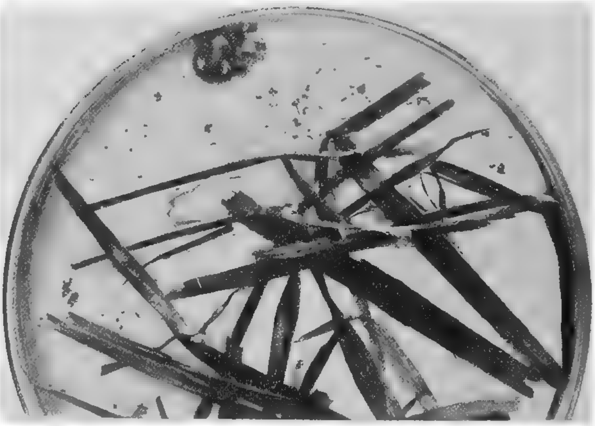


FIG. 54.—*Ixodes ricinus.* Female depositing eggs.

three or four millions. In severe cases the loss may be from one to two millions in the first twenty-four hours, and three or four millions the next day. The rapidity of the destruction is important so far as the colouring of the urine is concerned. If three or four millions are lost in five to eight days there may be no hæmoglobinuria, but if they are lost in one to two days hæmoglobinuria is certain to be set up. In the former case time has allowed the macro-organism to accommodate itself.

The duration of the disease varies, but the high temperature seldom lasts longer than a week to ten days. The fatal termination may take place in four or five days, at the height of the fever, or at a later stage, when it is probably due to exhaustion. Acute midsummer outbreaks are usually the most fatal.

As already pointed out, when recovery occurs the urine and temperature may both be suddenly restored to the normal condition. As the fever subsides the fæces become softer, and more or less bile-tinged. A varying period of debility remains, which may last from weeks to months.

The *mild type* is very likely to pass unobserved, and usually occurs in the later part of the year. Animals that have passed through an acute attack in the early part of the season may have a relapse in the form of a mild type in the autumn. When calves are affected, with a few exceptions, they usually develop this form. The main symptoms differ in degree from those of the acute type. There is slight dulness, loss of energy and condition, with diminished appetite. The respiration and pulse are accelerated, and the temperature may be elevated to about 104 to 105° F. The destruction of red blood corpuscles goes on very slowly, and consequently the discoloration of the urine may be slight or absent. The affected animals are at their worst for six or eight days and then recover, often with a long period of anæmia.

The *atypical case* is not uncommon, but in this country it is seldom diagnosed, since the chief symptom—hæmoglobin in the urine—is absent. In other respects the symptoms are almost identical with those of an acute attack, and it is not infrequently fatal. The invasion of the attack is comparatively slow, and the process of destruction of corpuscles and the liberation of the hæmoglobin is so prolonged that the whole of the latter is eliminated by the liver as in the normal process, and the kidneys are not called upon to take up the extra function. It is in these cases particularly that the name hæmoglobinuria becomes quite inadequate, and it would be better if the name bovine piroplasmosis were substituted for it.

Another atypical form is known as *Parturient Red-water*. This form occurs in animals that have been the subject of acute red-water and have developed tolerance still harbouring the parasites. The organisms have regained their virulence owing to the debility of the patient at the time of calving. The attack is usually very severe and is responsible for rather a high mortality.

Post-mortem Appearances.—Ticks may still be attached to the skin, and on reflecting the hide the small elevations previously noted as being due to tick punctures are seen to be somewhat hæmorrhagic. In very rapid cases the carcase may still be in fairly good condition, but in the more prolonged cases the carcase suffers, the flesh being paler than normal and the subcutaneous connective tissue œdematous, as is usually the case in emaciated and anæmic carcases. The fat is occasionally but not very frequently "jaundiced." The organs showing the most striking macroscopic alterations are the spleen and liver. The former is much enlarged, and may be from two to four times the normal weight. Its consistence varies and may be still firm, or semi-diffluent. On section the colour is darker red than normal, and the trabeculæ are indistinguishable. The liver is almost invariably three or four pounds heavier than normal, enlarged and congested; it is affected with fatty degeneration, showing greyish-yellow patches, and is very friable. The gall-bladder is usually distended with bile. The kidneys vary more or less in colour, the cortex being dark reddish-brown, or almost black, and friable in acute cases, while in prolonged cases the renal capillaries are filled with red corpuscles, most of them being infected with parasites. The bladder usually carries a quantity of urine which contains hæmoglobin and albumen in acute cases.

The alimentary tract often shows nothing abnormal. In those cases with diarrhœa there is often slight congestion of the intestinal mucous membrane, and sometimes a few small hæmorrhagic spots. In constipated cases the fæces are generally mucus-coated, and may be blood-stained. The peritoneal and thoracic cavities contain a little red-stained serous liquid. The lungs are very slightly affected; they are occasionally œdematous and may show pneumonic foci. The pericardium usually contains a small amount of reddish effusion. The right ventricle is distended with blood and the left almost empty. The epicardium and endocardium show ecchymoses of varying dimensions. The heart-muscle is often paler than normal, having a more or less macerated appearance. Its capillaries appear very injected. The brain usually shows no macroscopic alteration, but in those cases that have shown nervous symptoms the meninges are congested, and there is a little coloured effusion between them.

The Urine.—As pointed out previously, the special peculiarity of the urine is its red colour, which varies in intensity. The colour is not due to the discharge of blood from the kidneys and subsequent breaking up of the red corpuscles, but to the excretion of the hæmoglobin that is free in solution in the blood plasma, as the result of the destruction in considerable numbers of the red corpuscles in the general circulation (hæmoglobinæmia). The depth of the colour depends upon the proportion of hæmoglobin or methæmoglobin present. It may have a light claret colour, or it may have a colour somewhat like stout. The specific gravity is raised to 1030 or 1040 in the early stages but falls later. The reaction is either very slightly alkaline or slightly acid. It is distinctly albuminous, as is shown by the ordinary tests either of boiling or the careful addition of strong nitric acid. When boiled a brownish precipitate forms which rises to the surface as a scum. The addition of nitric acid causes a brownish, flaky precipitate.

Treatment.—Affected animals should be immediately removed from the pastures and placed in sheltered quarters with plenty of fresh air. All ticks found on the patient should be removed and destroyed, the search being frequently repeated to find those missed earlier on account of their small size. They are found principally on those parts of the body where the skin is thin (see *ante*). They are rarely present in such large numbers as to necessitate dipping, as they are in tropical countries. The daily application of creolin 5 per cent to the skin, particularly of the legs and the lower parts of the body, is sufficient to keep them in check. Medicinally, the use of trypanblue (introduced by Nuttall) has been found very effective in red-water due to *P. bigeminum* as in most cases of tropical red-water, and for that form may be regarded as a specific. The dose employed is grms. 1 to 2 in 1 per cent solution (*i.e.* 100 to 200 c.c.). This may be prepared approximately by dissolving 16 or 32 grains of trypanblue in 4 or 8 ounces of distilled water respectively. The solution may be injected intravenously or subcutaneously—the former preferably. This agent is apparently not so uniformly successful in British red-water due to *P. divergens*, which is probably the commoner form. In its place Stockman and Gibbings have employed antimony tartrate (tartar emetic) in doses of grs. xvi. dissolved in distilled water ʒii. (2 per cent solution) and injected into the jugular vein. Their published results, however, are not highly satisfactory.

In cases where there is constipation or the fæces are at all firm a full dose of a purgative, oleaginous or saline, should be given at once, and followed by stimulants and hæmatinics such as

℞ Carbonate of Iron,
Carbonate of Ammonia } āā ʒiii.
Powdered Nux Vomica, ʒi.
Ft. Pulv.
One to be given twice daily in cold gruel.

On account of the good results obtained from quinine in human malaria the same drug is often given to cattle, either by mouth or subcutaneously, but in this country it has not met with success. Many practitioners give turpentine with good results. Fowler's solution of arsenic is recommended by some, while if the heart is very weak, ether or camphor dissolved in ether injected hypodermically may improve its vigour.

In addition to the medicinal treatment the affected animals should be allowed plenty of fresh water and a laxative, nourishing, easily digested diet, such as linseed mashes, linseed cake, etc. Excitement should be avoided and shelter provided.

Preventive Measures.—Two methods of affording protection against the disease suggest themselves, directing one's attention either against the intermediary bearers of the organisms or against the organisms themselves. The disease can be considerably reduced by better cultivation of the land, better drainage, and by burning the rank, high grass and brush refused by cattle, but which afford protection to ticks, and from which the young ticks attach themselves to their victims. The application of crushed rock salt 10 cwt. to the acre also checks the development of ticks.

The application of parasiticides to animals grazing on infected pastures may check infection. (For practical measures against ticks see article on "Ticks.")

The exclusion of cattle for two winters and a summer will exterminate ticks from a pasture, since the larvæ developing from the eggs of the last ovigerous females would be unable to continue their development and would perish. On restocking care must be taken in order to avoid introducing fresh ticks. If stock from clean districts have to be imported into affected districts it is advisable, as far as possible, to do so while quite young, since the calves apparently possess a considerable degree of resistance. They may develop a mild attack followed by tolerance or by another mild attack the following year. If adults have to be transferred the change should be made in the late autumn or winter rather than in the hot weather, when the ticks may be very numerous and produce a severe attack. G. H. W.

Piroplasmosis of Sheep.—First investigated in Roumania, where the disease had been known for a number of years as Carceag of sheep. It has since been found in a good many tropical and sub-tropical countries—Southern France, Italy, Southern Russia, Turkey, West Indies, China, and various parts of the continent of Africa.

In Roumania the disease was found to occur mostly in damp pastures, seldom in mountainous districts, and cases were seen chiefly during the warmer period of the year. Native sheep were found to be much more resistant than imported ones. The parasite is known as *Babesia ovis*. It is very similar to *B. bigeminum*, but somewhat smaller, and usually occurs singly in the corpuscles; more seldom, double pear-shaped forms may be seen. After direct inoculation symptoms are first seen in from eight to ten days. Both adult sheep and lambs are affected, but the disease in lambs is usually much lighter. The transmitting tick in Roumania is the *Rhipicephalus bursa*, a dropping-off tick, but the larval and nymph stages of this tick are usually passed on the same sheep.

As in the case of the ticks that transmit canine piroplasmosis, the infection in these ticks does not become operative until they reach the adult sexually mature stage.

Symptoms are fever, loss of appetite, and profound anæmia. The number of corpuscles sinks from eight millions down to one and a half millions. Red-water is often seen, the urine becoming coffee-coloured. The sheep are often affected with diarrhœa and the fæces are tinged with blood. The symptoms may run their course in from two to three days, and the mortality amounts to about 50 per cent.

On post-mortem examination the appearances are very much the same as in other piroplasmoses. Spleen is very much enlarged. There is an œdematous infiltration of subcutaneous tissues and the serous membranes due to heart failure. In the intestine one finds signs of catarrh or even of an ulcerative enteritis. The liver is affected with cloudy swelling. J. T. E.

Piroplasmosis of the Dog. Malignant jaundice.—Occurs in Southern Europe, Africa generally, and in Asia. Caused by *Piroplasma* (or *Babesia*) *canis*, an organism which resembles *Piroplasma bigeminum* very closely but differs from it in that multiple invasion of a single corpuscle may take place (up to ten parasites may be seen in one corpuscle). Ring-forms are rather more common than in *P. bigeminum*. Multiplication takes place by budding and by simple fission. The transmitting tick differs in various countries. In South Africa the tick is the *Hœmaphysalis leachi*; in Asia and North Africa, *Rhipicephalus sanguineus*; in France, *Dermacentor reticulatus*. These are all dropping-off ticks. Transmission appears to take place in all in the same manner. The tick becomes infected as the adult female tick, and its progeny does not become infective again until it reaches the succeeding adult stage (Brumpt). The larvæ and nymphs cannot be infected. The larvæ and nymphs obtained from infected females may be fed on non-susceptible animals, such as the hedgehog or rabbit, and still be capable of infecting a susceptible dog on arrival at the adult stage again. The development of the dog piroplasm in *Rhipicephalus sanguineus* appears to be as follows according to Christophers.

Development begins in sexually mature ticks. A considerable increase in size of the parasite takes place in intestine. It throws off a part of its protoplasm, grows again considerably, forms club-shaped bodies. Probably some of these are male forms and other females, but fertilization has not actually been observed. These club-shaped bodies are actively motile, penetrate through the stomach wall, and wander about into all the tissues including the ovaries and the eggs. This stage would therefore correspond with the oökinete of the malarial parasite. In the ovary it becomes converted into a larger round cell up to about 25 μ in diameter, and within it the chromatin is seen either as a single compact mass or divided into a number of small fragments. Division into sporoblasts takes place subsequently in the tissues of the larval ticks or in the organs of the nymphs. Each time the tick moults pronounced changes take place in its tissues and thus the sporoblasts become distributed throughout its body. Each sporoblast then divides up into 50 sporozoites, which migrate to salivary glands and are there very similar to piroplasms found in the blood. The cycle is thus practically complete, but it is not really completed till the tick reaches the late nymph or the commencing adult stage.

The number of piroplasms discoverable in the blood varies. On the third or fourth day of symptoms one in ten of the red corpuscles is invaded.

Symptoms.—These may be classed as acute or chronic. In acute form the incubation period is usually from seven to ten days. After natural transmission by ticks it extends to from thirteen to twenty-one days. The virulence of the organism may be exalted by passage until the period is reduced to from three to four days. The affected dog is dull, refuses food, and exhibits great thirst. Young dogs show diarrhœa, which is frequently watery. The patient shows weakness in the hind quarters, and this weakness extends until the whole body becomes paralysed. In the most acute form the dog may be found dead in eighteen hours, but the acute form usually lasts from four to ten days. Post-mortem — the chief signs are those of acute anæmia. In two or three days the number of red corpuscles may sink from six to seven million down to two million per cubic millimetre. As is usual in anæmia, the number of leucocytes is simultaneously increased so that they may rise from 7000 up to 40,000 per c.mm. The mucous membrane assumes a cyanosed colour. Hæmoglobinuria is seen usually in about two-thirds of cases. In cases that last just a little longer a yellowish or jaundiced discoloration of the mucous membranes is very noticeable. White-haired dogs may actually assume a golden yellow colour. In animals which are dead after about four days the spleen enlargement is marked.

In chronic cases the symptoms are of the same order but signs of liver disorder are more pronounced. The dog shows signs of jaundice, extreme debility, and wasting. These cases last about a fortnight or a little longer, and then the dog dies from exhaustion or very slowly recovers. In countries in which this disease is enzoötic the more acute symptoms are usually seen in dogs, such as sporting dogs, imported from non-infected countries. Native-bred dogs show a much higher degree of resistance.

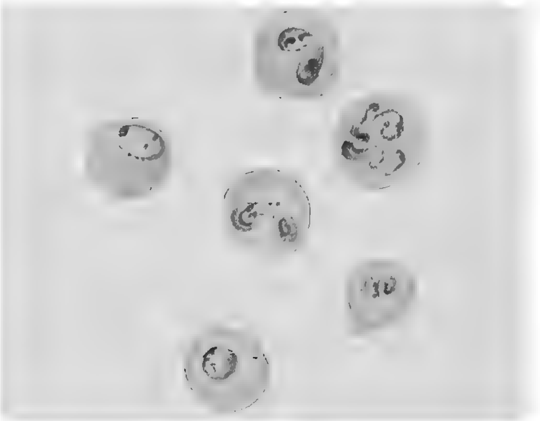


FIG. 55.—*Piroplasma canis.* (After Schilling.)

In chronic cases on post-mortem examination the spleen enlargement may not be noticeable. Subcutaneous tissues and serous and mucous membranes are deeply tinged yellow.

Treatment.—Nuttall proved that trypanblue is a remarkable specific. The dose for a dog of average size is 5 c.c. of a 1 per cent solution. In six hours after injection the piroplasms are greatly deformed, and a few hours later they seem to disappear completely from the blood. Relapses are apt to occur, and recovered dogs have been proved to have harboured the piroplasms in their blood for at least two and a half years.

It has been shown that in cases where it is very difficult to demonstrate the parasite in the peripheral blood they can be demonstrated with greater ease in the capillaries of the deeper organs such as the brain and kidneys and spleen, especially spleen and kidneys. Some workers think that more than one strain of organism exists. The South European strain is less severe than the African. J. T. E.

Equine Piroplasmosis. Biliary Fever.—First found in Italy, but it has since been found of very common occurrence in most tropical and sub-tropical countries, chiefly South Africa, India, Southern Russia, and South-Eastern Europe, especially in the Balkans.

The parasite, which is seldom seen in the blood in large numbers but is more numerous in the spleen and other deeper tissues, was until recently considered to be of one type. A number of recent observations, however, have been made to prove that biliary fever may be set up by two different types of parasite, viz.: (1) *Piroplasma* (or *Babesia*) *caballi*, and (2) *Nuttallia equi*.

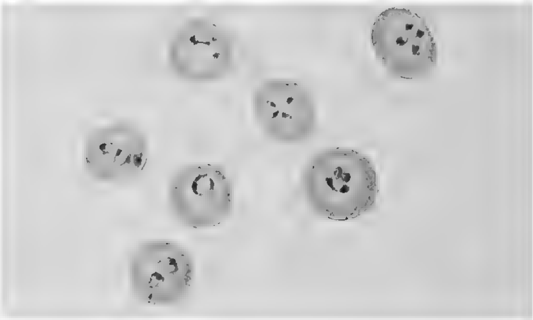


FIG. 56.—*Piroplasma equi.* (*Nuttallia equi.*) (After Schilling.)

P. caballi is very similar to *P. bigeminum.* It usually is large, rounded, and frequently pear-shaped. Double pear-shaped forms are frequently seen.

The Nuttallia parasite is smaller. To commence with, the parasites that appear in the blood are similar in shape to the piroplasma parasites, but in two or three days forms are seen which exhibit a peculiar method of division. Within a red corpuscle there are seen four daughter parasites arranged radially so as to form a pattern like that of a Maltese cross. Each element is usually considerably smaller than those of the *P. caballi* type.

After the disappearance of the acute symptoms it is usually impossible to find the parasites in the peripheral blood. These diseases (*i.e.* that due to *P. caballi*, and that due to *N. equi*) are frequently found in close association, *i.e.* they may occur in the same district and a horse may be infected with both types. The *caballi* infection is as a rule seen earlier in the year than the *equi* infection. In districts where the *caballi* infection alone exists cases are more frequently seen in April and May. In countries such as India, where *Nuttallia equi* infection predominates, cases may be seen throughout the warmer part of the year. This difference in seasonal distribution is due to the fact that the parasites are transmitted by different species of ticks. In the Balkans it has been shown that *P. caballi* is transmitted by *Dermacentor reticulatus*, while *N. equi* was transmitted by *Rhipicephalus bursa*. In Southern Russia, where the *caballi* infection occurs alone, the tick is the *Dermacentor reticulatus*. In South Africa the transmitting tick was found to be *Rhipicephalus evertsi*, or red-legged tick. This latter tick passes its larval and nymph stages on one host, falls to the ground after from thirteen to twenty days, and after moulting seeks a new host (horse or ox). The disease is transmitted, as in the case of carried infection, only by sexually adult ticks. It is passed on through larva and nymph. It has been proved recently that the common European tick, *Ixodes ricinus*, cannot transmit the *N. equi* infection.

Symptoms.—Usually sudden in their onset. The incubation period varies. Naturally transmitted by the tick, it extends up to about three weeks, but by artificial inoculation the period is much shorter, and after a number of passages through the horse it may become shortened to about three days. Two forms have been recognized, viz. an acute form which is usually fatal, and a mild form.

In the acute form the symptoms are those of jaundice, which appears very early (it has been suggested that this is due to the horse having no gall-bladder). There are also symptoms of heart weakness and alternating diarrhœa and constipation. This form lasts from three to seven days. The horse suddenly collapses, temperature becomes sub-normal, the urine is yellow, and hæmoglobin is very seldom found in it. The mortality in this form varies from 30 to 80 per cent.

In the mild and most commonly occurring form the symptoms at first are also somewhat sudden in onset and rather acute. Jaundice and slight fever are marked symptoms, together with pronounced anæmia. The corpuscles may decrease to a quarter of the original number. The animal appears dull and depressed, and is affected with œdematous swellings of the dependent parts, especially of the hind extremities; wasting is marked. If there are no complications the mortality from this form is slight, usually not more than 5 per cent, and the disease may be confused with an ordinary attack of equine influenza.

If affected horses, however, are kept at work and not rested immediately on showing symptoms, the mortality may become considerable. A disease with which this is likely to be confused in certain countries is equine pernicious anæmia. All breeds of horses are susceptible, but in countries where the disease exists native horses show a high degree of resistance. They are undoubtedly bitten by transmitting ticks as foals and acquire thereby a high degree of so-called immunity. The parasite has been proved, however, to remain in the blood for a considerable number of years and the animals act as carriers. Imported horses suffer much more severely.

Treatment.—Various forms of drug treatment have been recommended, chiefly intravenous injection of quinine, biniodide of mercury, and arsenical compounds. It has been proved, however, that they have no effect. Trypanblue has no effect on *Nuttallia equi*.

Prophylactic measures comprise avoidance of tick-infected pastures, and especially shrubby ones, during the tick season. J. T. E.

Piroplasma mutans

It was found that when imported animals were injected in South Africa with blood from native cattle, two or three separate rises of temperature could be subsequently observed: (1) a rise in from eight to ten days due to *P. bigeminum*; (2) a rise in from twenty-six to twenty-eight days due to some other parasite, which has now been found to be *Anaplasma marginale*, and then (3) a third rise in forty days due to *P. mutans*. The incubation period for *P. mutans* has, however, been found to vary from twenty to forty-five days. A slight temperature is observed for about a week. The animal has a staring coat. The appetite is diminished, there is some loss of condition, but there is never any red-water, and there are never any Koch's blue bodies in the lymphatic tissue. Nearly all cattle in South Africa are affected with this piroplasm and it appears to produce no ill effect, but a relapse of infection may be produced when an animal becomes infected with other diseases, such as heartwater or pneumonia. The parasite is transmissible by blood inoculation, and is probably transmitted naturally by the same species of ticks as ordinary red-water. J. T. E.

EAST COAST FEVER

East African Coast Fever; African Coast Fever; Rhodesian Red-water; Rhodesian Tick Fever; Tropical Piroplasmosis; Bovine Theileriasis

Definition.—An acute specific disease affecting bovine animals, caused by the *Theileria parva*, and transmitted by ticks belonging to the species Rhipicephalidæ.

History.—This disease was first investigated by Koch in German East Africa in 1897, who regarded it as a form of Red-water; but it was not until 1901, when it was introduced into Southern Rhodesia with a consignment of cattle shipped from Dar-es-Salaam for Beira and Delagoa, that it attracted general attention. It is alleged that on the voyage south a large number of these cattle died and the carcases were thrown overboard. Some of the survivors were landed at Beira and the ship went on to Delagoa Bay. Of the lot landed at Beira, several truck-loads were sent by rail to Umtali, and two loads went on to Salisbury. The disease broke out among both lots, but its true nature was not recognized. In an official Gazette, No. 135 of 1902, it was stated that the cumulative positive and negative evidence left no reasonable doubt in the minds of any of the qualified and professional men who took part in the investigations as to the disease being certainly Bovine Malaria or "Red-water." In this manner the misleading and undesirable name "Rhodesian Red-water" originated, which in the light of existing knowledge should be discarded. From the original centres the disease extended over the main transport roads and thence to the farms adjoining the roads. During the first few years the losses among stock owned by Europeans were enormous, and it was estimated that some 95 per cent of all infected animals died. No remedies were found of any avail, and to this day no successful curative agent has been discovered. During the years 1904 and 1905 the disease abated, probably by reason of the fact that most of the cattle on the infected areas had succumbed. Since then the veterinary measures, based upon a more scientific knowledge, have been successful in limiting the disease to a few isolated outbreaks.

The disease invaded the Transvaal in May 1902, but its arrival was disguised by the presence of Rinderpest which existed at that time. As in the case of Rhodesia it was probably introduced by cattle imported from German East Africa; no connection can be traced to the outbreaks in Rhodesia.

In many parts of Central Africa the disease is enzoötic, its manifestation depending upon local conditions. Thus in certain areas large numbers of cattle exist in spite of the prevalence of the disease, its existence being shown by the immunity of survivors when moved from such areas and exposed to infection elsewhere. It is possible that the virulence of the disease is attenuated by passage through cattle bred upon such areas. In other districts where large parts of the country are available for grazing, enzoötic areas are detected by the very heavy mortality of calves and can be avoided by removal to new pastures, so that immunity is not established in the herds which escape infection.

A disease affecting calves in Uganda, and known as "Amakebe," was investigated by Bruce and found by him to be an enzoötic form of East Coast Fever.

Cause of the Disease.—African Coast Fever is essentially a disease of bovine animals, and is caused by a minute parasite (*Theileria parva*), which undergoes development partly in the tick and partly in the ox. Having been taken up by the tick from an infected animal, it undergoes sexual multiplication, giving rise to certain spore-like bodies which are inoculated by the tick into the animal it bites. These first establish themselves in certain organs, such as the lymphatic glands and spleen, where they invade the white blood-cells, in which they undergo non-sexual division, producing the so-called "Koch's bodies." These break up into a number of small parasites which invade

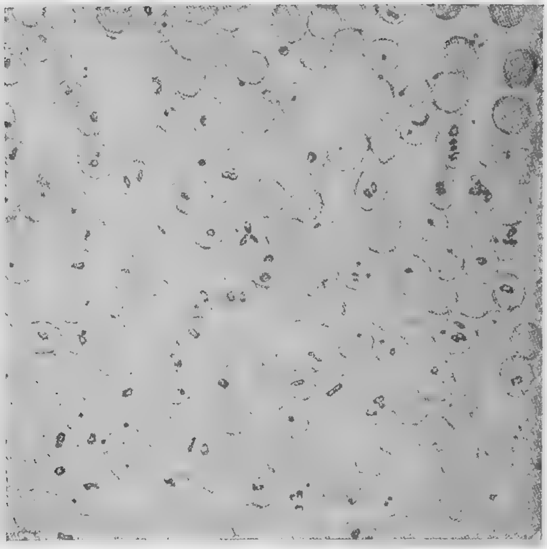


Fig. 57.—Blood film, showing parasites in the red cells.

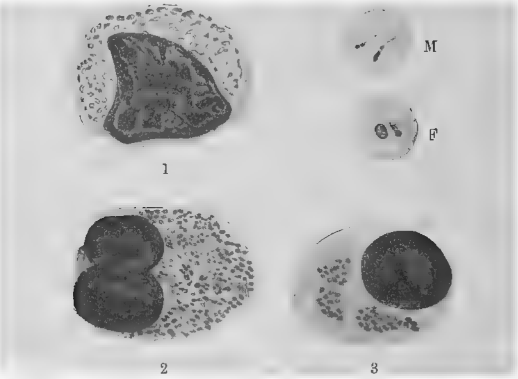


Fig. 58.—*Theileria parva*. (After K. F. Meyer.)

M, Male gametocytes in red corpuscle; F, female gametocytes in red corpuscle; 1, 2, and 3, Koch's blue bodies in lymphoid cells; 1, large multinuclear agamonts; 2 and 3, multinuclear gamonts in mononuclear cells.

the red corpuscles, with which they are taken up by the ticks feeding upon the ox at that time, and in certain species of ticks undergo further development. At the height of the disease in the ox, some 75 per cent of the red corpuscles are invaded, but since there is little reduction in the number of the red cells it is assumed that the parasite does not bring about their destruction, although it may obtain nourishment from them. It is also probable that the principal injury to the host is caused by the toxic materials produced by the parasite.

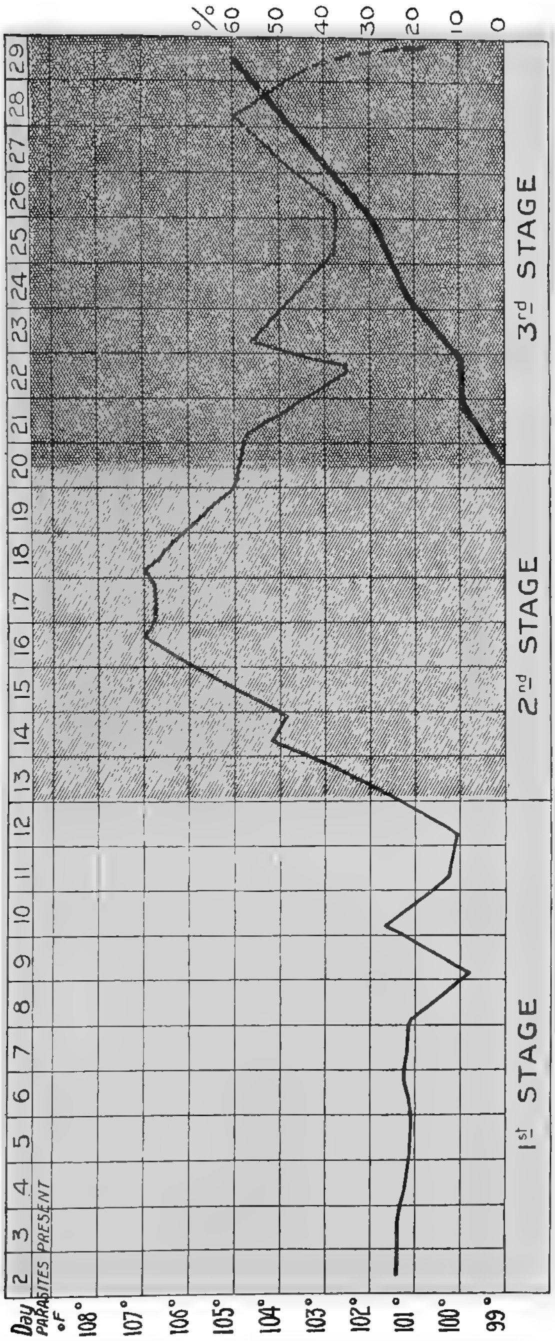


FIG. 59.—Characteristic chart of African Coast Fever in a cow infected in England by ticks sent from South Africa. The thin black line indicates the temperature record. The thick black line indicates the rise in the percentage of infected red blood cells.

Stage 1. The period of incubation. Stage 2. The period when the temperature is elevated, but parasites not present in the blood. "Koch's bodies" may be found in the spleen and glands. Stage 3. The period when parasites are present in the peripheral blood.

Although numerous parasites may be present in the blood, the disease cannot be transmitted from an infected to a susceptible animal by blood inoculation from one to the other. The reason is that the parasites in the blood are non-sexual forms and incapable of undergoing further development in the ox. It is believed that if an animal recovers, the particular parasites in it die out, and it is on these grounds that the "salted" animal is assumed to be incapable of perpetuating the disease.

Incubation and Course of the Disease.—From the time the tick introduces the parasite into a susceptible animal, an interval of from ten to twelve days elapses before the first manifestation of the disease, namely, a rise in temperature. About this time, or shortly after, the so-called "Koch's bodies" can be detected in the glands and spleen. A further period of from three to eight days elapses before parasites appear in the blood, during which time the animal becomes visibly sick. The number of cells invaded by these parasites increases daily, and towards the end of the disease up to 75 per cent may be involved. The average time from the day of infection until death is about thirty days.

The shaded diagram given, which may be taken as representing the usual course of the disease, may help to make this explanation clear. In it the unshaded portion represents the first stage, when the disease cannot be detected by the microscope or by clinical observation; the lightly shaded portion is the second stage, when the temperature is elevated and "Koch's bodies" are present, but no parasites in the blood; the darkly shaded portion represents the third stage, when parasites are present in the blood and can be taken up by ticks feeding upon the animal. Thus the third stage is the period of danger.

Although the chart is typical of the majority of cases and can be taken as a basis upon which to apply the preventive measures about to be described, cases running an unusual course are frequently met with. For example, in a herd under careful observation where the temperatures are regularly taken, cases are met with in which the temperature rises, but returns to normal after a few days.

In these cases a relapse may occur after several weeks and the animal may die; or there may be no relapse and the animal may recover. Such cases have been more numerous since systematic dipping has been practised as a method of suppressing the disease. It has been shown by experience that in animals habituated to arsenic by constant dipping, the infective processes are retarded and the average period of incubation may be extended to sixteen days, but it is now clear that it exerts other influences upon the course of the disease which cannot be fully explained.

Symptoms of the Disease.—The first indication is a sudden rise of temperature, which may reach 107° F.; but, notwithstanding the fever, outward indications are rarely met with before the last few days preceding death. These are not very distinctive, but the sick animal may evince one or more of the following symptoms: dulness; falling-in of the flanks; running from nose, eyes and mouth; a husky cough; swelling of the glands; weakness of the loins; staggering gait and tendency to lag behind the herd; constipation, followed by diarrhœa; and there may be blood in the fæces. The tissues around the eyes may be swollen and the eyes appear sunken. Frequently the sick animal will continue to feed until the disease is advanced; finally, however, rumination ceases and appetite is lost. When the lungs are involved, the animal will grunt when walking downhill, and there may be distress in breathing.

Post-mortem Appearances.—The most important lesions are: yellow discoloration of the fat; enlargement and congestion of the glands, especially of the throat and neck; alteration in the blood, indicated by small blood spots or hæmorrhages on the surface or lining of the heart, on the serous membranes and in the gall-sac and bladder. In a great many cases the lungs are affected, being dark in colour and infiltrated with straw-coloured fluid, which collects between the lobules or escapes into the chest cavity. This material in the air-passages becomes whipped up into a foam, which passes up the trachea and escapes by the nostrils, as in the photograph. The lining of the fourth stomach or abomasum is inflamed and frequently ulcerated; the blood spots are met with here and throughout the intestines. Small white, yellow or purple spots varying in size from a pin's head to a pea, known as infarcts, are seen as small elevations or depressions on the surface of the liver and kidneys. When cut into, they are generally triangular or conical in shape, the base being on the surface and the apex in the depth of the organ. These infarcts, when present, are almost diagnostic.

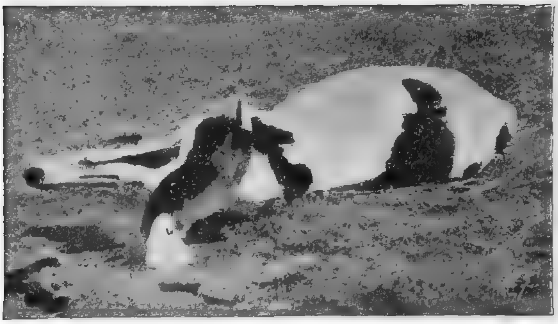


Fig. 60.—Animal dead of East Coast Fever showing froth at the nostrils.

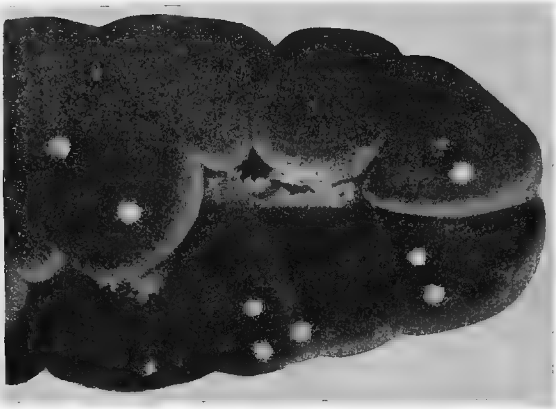


Fig. 61.—Portion of kidney from a case of African Coast Fever showing areas of necrosis (infarcts) in various stages. (From pamphlet by W. Robertson, M.R.C.V.S.)

Transmission of the Disease.—African Coast Fever is not directly contagious; a sick animal will not infect another susceptible animal by contact or by the excretions or fluids of its body. The disease is only transmitted by the bite of certain species of ticks, namely, *Rhipicephalus appendiculatus*, or brown tick; *Rhipicephalus evertsi*, or red-legged tick; *Rhipicephalus simus*, or black-pitted tick; *Rhipicephalus capensis*, or Cape tick; *Rhipicephalus nitens*, or shiny-brown tick. Of these, the brown tick is the chief offender, and methods of controlling the disease are principally based upon a knowledge of its habits.

There are four stages in the life-cycle of ticks: (1) eggs; (2) larvæ or seed ticks; (3) nymphs; (4) adults or sexual ticks.

In the case of the brown tick, the adult female, having become engorged and fertilized, falls to the ground, where she finds a suitable place, such as the loose soil in kraals, and deposits some three to five thousand eggs. In from five weeks to five months—according to climatic conditions—the larvæ or "seed ticks" hatch out. These may remain unfed for several weeks until a suitable host is available, upon which they may remain attached for three to ten days. Having fed, they fall to the ground, and moulting in about sixteen days become nymphæ, and can remain alive unfed for several months, but will then attach and feed, remaining on the animal from three to ten days. Having engorged, they fall to the ground, and in about eighteen days moult and are then ready to attach to a suitable host to feed as adults, although they may survive many months unfed. Having attached, they remain on the host from four to ten days, during which time they feed and copulate. Although larvæ and nymphs readily attach during the cold weather, the adults are scarce on cattle during the winter. Unfed adults have been known to live 630 days under laboratory conditions, and in practice it has been found that a race of infected brown ticks on a farm from which bovine animals have been removed may survive as long as fourteen months, possibly longer, and still prove infective.

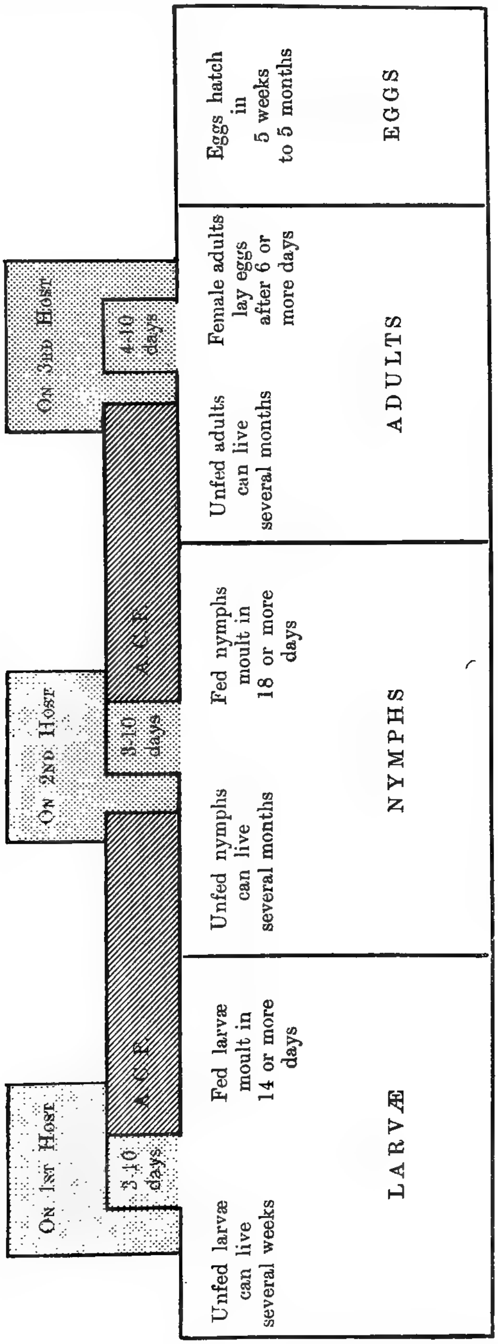

Eggs hatch in 5 weeks to 5 months
EGGS
Female adults lay eggs after 6 or more days
Unfed adults can live several months
ADULTS
Fed nymphs moult in 18 or more days
Unfed nymphs can live several months
NYMPHS
Fed larvæ moult in 14 or more days
Unfed larvæ can live several weeks
LARVÆ
On 3rd Host
4-10 days
A.C.F.
On 2nd Host
3-10 days
A.C.F.
On 1st Host
3-10 days

Fig. 62.—The Brown Tick (*Rhipicephalus appendiculatus*).

While the brown tick requires three hosts upon which to feed in its different stages, the red-legged tick requires only two, since the larvæ crawl into the ears of their hosts, where they feed and moult and feed again as nymphs, remaining on the animal about fifteen days.

It is fortunate that the parasite of African Coast Fever does not pass through the egg stage, as in the case with the parasite of red-water. However, if a larval brown tick has engorged on an infected bovine animal having the parasite of African Coast Fever in its blood, it can infect a susceptible ox to which it attaches as a nymph; or, again, if a nymphal brown tick has fed on an infected animal, it can infect a susceptible ox to which it attaches as an adult.

African Coast Fever is peculiar to bovines, and the causal parasite cannot establish itself in any other species; so that ticks can only obtain the parasite from an infected bovine animal, and then only from one in the last stages of the disease, when the parasites are present in its blood. If, therefore, an infected tick bites and completes its feed upon a horse, sheep or other non-susceptible animal, the parasite is discharged without affecting the animal, and the tick thereafter becomes non-infective. But it has been found that infected ticks are still infective after feeding upon a rabbit for three days, if they complete their feed upon a susceptible animal within seventeen days.

Infected ticks may, however, be carried by other animals to which they do not attach—by human beings in their clothing, in hay or crops, in the hides (especially the ears) of dead animals—and may thus be transported many miles. Precautions must always be taken against such risks, although in practice the great majority of outbreaks are brought about by ticks which have fallen directly from an infected ox which has at some previous date passed over or died in the vicinity.

The foregoing details may be more easily understood from the diagram. They are of considerable importance, because upon them are based the principles for the control of the disease.

How to determine the Source of an Outbreak.—The disease having broken out, it is generally necessary to determine whether the animals became infected on the farm or picked up infected ticks from without. In coming to any conclusion, it should be borne in mind that: (*a*) If the sick animal has been on the farm for a time covering the period of incubation, or roughly, a fortnight before showing a rise in temperature, or one month before death, it is practically certain that it contracted the disease on the farm. (*b*) If, however, the disease appears in animals which have not been on the property longer than a week, it is probable that it was contracted before they arrived; and in endeavouring to ascertain where they became infected, it should be remembered that the disease may be in the system for about three weeks before visible symptoms are manifest, and that roughly one month elapses between the time of infection and the death of the animal. (*c*) It frequently happens that the first case of a disease is overlooked, and that it is only diagnosed when quite a number of animals are affected at or about the same time—what is generally known as the "second crop" of cases. During the last week of its life, the first animal to contract the disease supplies blood containing the parasite to those brown ticks feeding upon it. The larval or nymphal ticks dropping during that week seek shelter, moult, and when the opportunity arises, re-attach. As explained, this process takes at least sixteen days and generally longer, although the nymphs and adults may survive unfed several months awaiting the arrival of a suitable host. In addition to this, the disease is not noticeable in an infected animal until the last stages of the disease, often about twenty-one days; so that the "second crop" of cases does not generally occur until at least six weeks after the first case. It may happen that, if many ticks survive, quite a considerable number of animals become sick about the same time; but one tick is sufficient to carry on infection, and laboratory experiments show that an adult tick can survive unfed as long as twenty-one months. Therefore, the alarming and original theories so frequently advanced to explain outbreaks, where previous movements of cattle cannot be fully traced, are generally unnecessary.

Precautionary Measures.—(1) *Short-Interval Dipping.* African Coast Fever is a disease which can easily be prevented by the simple expedient of systematic dipping. If introduced to a farm where short-interval dipping is practised the disease cannot establish itself; and, when established on a farm, it can be quickly eliminated by the application of the process.

The term "systematic" or "short-interval" dipping implies the immersion of cattle and other domestic animals in solutions destructive to ticks and other parasites, at certain intervals based upon a knowledge of the life-cycle and habits of these parasites. In the case of African Coast Fever, the principal object to be attained is the destruction of the brown tick, and this presents certain difficulties because, as we have seen, this tick seeks different hosts in the larval, nymphal, and adult stages, and may escape from them in as short a time as three days. Therefore, if it is to be caught, the dipping must be carried on at short intervals, and the dipping solution must be of such strength that the cattle can be immersed in it as frequently as every third day without being harmed by it. To obtain a dip thus harmless to cattle, and at the same time destructive to the tick, was a matter of considerable difficulty, but the problem was finally solved by Watkins-Pitchford, who, after numerous experiments, evolved the so-called "laboratory dip," which was made up of

5½ lbs. soft soap,
2 gallons paraffin,
8½ lbs. arsenite of soda (80 per cent arsenic),
400 gallons water.

But when it was found that certain ticks could attach and escape within as short a period as seventy-two hours, the composition was modified to form what was known as the "three-day dip," namely:

3 lbs. soft soap,
1 gallon paraffin,
4 lbs. arsenite of soda (80 per cent arsenic),
400 gallons water.

Since Watkins-Pitchford's discovery, several proprietary dips of a similar composition, and based upon the same principles, have been put on the market.

In addition to the actual in-contact effect upon the ticks, it is found that these solutions protect the animal for a short period by their repellent effect, and also by reason of the fact that a certain quantity of arsenic is absorbed through the skin and permeates its lower layers and becomes stored there; so that it is probable that the tick also becomes poisoned by the ingestion of arsenic.

Dipping fluids should be frequently tested. In hot weather they are liable to become too strong, as the result of evaporation; in the wet weather too weak, as the result of flooding. A solution under strength is a serious danger, by giving rise to a false sense of security.

It is a habit of the brown tick and the red-legged tick to make their way into the ears and attach themselves to the eyelids, where they may remain protected from the dip, as animals which are accustomed to being dipped learn to avoid the complete immersion of the head. Ticks under the root of the tail often escape owing to some animals keeping the tail compressed when being dipped; others in the tuft of the tail are also protected by the matting of the hair. Therefore, if the best results are desired in combating African Coast Fever, these parts should receive special dressing in addition to the dipping, for it is most important that no infected tick shall escape. For this purpose a mixture made by dissolving six to eight paraffin wax candles in one quart of paraffin oil has been recommended.

Both the blue and the brown tick will attach themselves to equines, sheep, goats, and other animals, but in so doing to a great extent lose their infectivity to cattle. Nevertheless, a farm on which ticks are few is better ensured against disease than where they are plentiful, and any system of dipping should, as far as possible, include all classes of stock on the farm. Again, it must be borne in mind that in various stages the ticks can remain for long periods on the ground without feeding, and any system of dipping must be carried out regularly and for a considerable period, to ensure that the crops of ticks are caught as they attach. A dipping campaign must therefore extend over a lengthy period.

By systematic dipping, cattle and other animals act as tick traps, collecting the ticks and conveying them to the dip to be destroyed; and, if the process is conscientiously carried out, the desired results are often achieved even more quickly than might be expected. In one of Watkins-Pitchford's experiments it was found that a badly infected area was cleansed of brown ticks in as short a period as three months. Apart from the protection afforded against African Coast Fever, the benefits to be derived from systematic dipping in eradicating red-water and allied diseases, and thus bringing about a general improvement in his stock, compensate the farmer for the trouble involved. The importance of regular dipping is no longer based upon theory, but has been abundantly proved by practical experience.

(2) *Temperature-Camp System.*—In the days before short-interval dipping was introduced, African Coast Fever was often successfully dealt with on the temperature-camp system. This was based upon the knowledge that the infected animal showed an elevated temperature for several days before the parasites made their way into the blood and the ox became a source of danger. That is to say, advantage was taken of the period of warning, which in the chart is indicated by the light shading and is called the "second stage." For the success of the system it was necessary that there should be certain clean veld to which cattle could be shifted. This was divided into a small clean temporary camp (Camp No. 1) and a larger clean permanent camp (Camp No. 2). A crush-pen having been erected, the temperatures of all in-contact animals were taken daily. Those showing an abnormally high temperature were marked, and if this continued for a second day were isolated or destroyed. With oxen and animals of average size and breeding, an early morning temperature of 103° F. was regarded as suspicious. With calves and highly bred or nervous animals, a temperature of a little above the average was to be expected, and a certain amount of discretion had to be exercised in interpreting the readings. It was rather upon a sequence of high temperatures than a single elevation that animals were condemned, and, when possible, the temperature reading was controlled by microscopic examination of gland smears in which from infected animals "Koch's bodies" could be detected before the animals became a source of infection. Infected animals having been detected and disposed of, all in-contact animals were carefully sprayed and were moved on to Camp No. 1, and were there "temperatured" daily for at least three weeks. If during that time any suspicious rise of temperature persisted for two or more days, they were destroyed or sent back to infected veld.

Those animals which registered a normal temperature throughout the three weeks were again carefully sprayed and passed on to the second camp, where they remained unless a further case occurred, which indicated that Camp No. 2 was not clean veld; in which case the process had to be gone through again, and the cattle were once more moved on through temperature camps to new grazing thought to be clean.

The foregoing method is even now resorted to in dealing with an outbreak where dipping tanks are not available, and is generally combined with short-interval dipping when dealing with serious outbreaks, the extent of which is not understood. The closer settlement of the land, however, has generally limited the areas to which in-contact animals can be removed. With this system, infected pastures had to remain free from cattle for a minimum period of fifteen months to allow the infected race of ticks to die out, whereas with the short-interval dipping, by the collection and destruction of ticks, the veld becomes cleaned in a far shorter time. The latter system, therefore, is quicker and far more satisfactory and has generally replaced the temperature-camp system.

LL. E. W. B.

ANAPLASMOSIS

Gall-sickness

Anaplasms were first discovered in 1893 by Smith and Kilborne in their investigations on Texas fever, but these workers considered the bodies to be in the nature of degenerated piroplasms. They were later discovered in 1904 in South America by Kolle, but the chief source of information on this question is Theiler in South Africa and Lignières in Argentina. The anaplasms take the form of coccus-like bodies from 0·1 to 0·6 of a micron in diameter, and they are usually placed towards the periphery of a red blood corpuscle. On this account they were originally termed marginal points by earlier workers, and the name now given is *Anaplasma marginale.* Very occasionally they are disposed towards the centre of the corpuscle. On account of the fact that the disease produced by centrally placed anaplasms is of a milder character Theiler gave the name *Anaplasma centrale* to this form, which he believed to be a variety of the other form. As a rule from 2 to 15 per cent of the blood corpuscles are invaded, but exceptionally as many as 50 per cent. The parasites apparently produce severe blood destruction. The usual anæmic blood changes can be well seen in from six to eight days after the commencement of symptoms. Many workers of repute, however, have disputed the protozoan nature of these bodies. They hold them to be either in the nature of degenerate piroplasms such as are frequently seen in blood severely infected with *P. bigeminum*, or on the other hand they may be of the nature of protoplasmic degenerations in the blood corpuscles themselves, such as are seen in cases of acute anæmia produced by any method. The protoplasmic degenerations seen in anæmic blood are known as Jolly bodies. They are seen in many species of animals normally or when affected with some other disease. The case for the protozoan nature of the so-called anaplasms is, on the other hand, very strong. If an ox is injected with red-water blood in England before exportation to South Africa, then very often after arrival in that country it shows in from twenty-three to twenty-five days high fever together with the presence of marginal points in the red corpuscles. If, on the other hand, the animal is exported to South Africa and submitted to inoculation in that country with red-water blood, then it shows a typical red-water reaction after eight to ten days, and from ten to twenty days afterwards a second reaction with the presence of anaplasms in the blood. Moreover, cases of pure infection with anaplasms are stated to have been found.

Transmission.—Anaplasms can be transmitted by direct inoculation, and the period of incubation is from twenty-five to twenty-eight days. Native South African cattle are much less susceptible than imported English cattle. Calves are much less susceptible than adults, as in the case in red-water. In infected countries cattle become infected as calves, and subsequently develop a high degree of immunity. The disease is not transmitted by inoculation of filtered blood. Quite recently Lignières has claimed that sheep and goats are susceptible to infection (thus the anaplasms differ from piroplasms, which are highly specific), but the anaplasm injection does not set up in these animals any symptoms except perhaps very slight fever from twenty to thirty days after inoculation. The animals can, however, be shown to have been actually infected by taking some of their blood and inoculating a susceptible animal. A typical anaplasmosis reaction is then set up in the ox. Lignières claims that the virus can thus be transmitted from sheep to sheep and goat to goat in series an indefinite number of times, that the virus is harboured in the body of the sheep and in the goat for a very considerable time, and that during this passage the virus undergoes a distinct degree of attenuation.

In countries where anaplasmosis exists it is seldom seen as a pure infection but is nearly always associated with a red-water infection. By passage through the bodies of sheep and goats the mixed infection can be cleansed of the piroplasms and pure anaplasm material obtained for vaccination. The disease is naturally transmitted by ticks. In South Africa the blue ticks, *Boöphilus decoloratus*, and black tick, *Rhipicephalus simus*, have been incriminated. The incubation period after tick infection may extend to seventy-two days, and a very mild type of the disease is produced.

Symptoms.—In adult cattle the symptoms are usually severe and the disease often ends fatally. They are loss of appetite, rapid wasting, skin becomes dry and yellowish in unpigmented places, the conjunctival and buccal mucous membranes are tinged yellow; sometimes there are œdematous swellings of the dependent parts. There is salivation, at first constipation, followed by diarrhœa. The urine is usually of a dark yellow colour, rich in bile salts, but it never contains blood pigment. Before death there are muscular weakness and paralysis. In animals that eventually recover the period of convalescence is very long. The mortality amounts to from 10 to 20 per cent. In some outbreaks 50 per cent has been recorded. Recovered animals remain reservoirs of the virus. After an attack an animal develops a considerable degree of immunity, which, however, is not so great as that acquired after one attack of red-water. The animal may even suffer a second attack, but this is usually mild.

Preventive measures are identical with those recommended for Texas fever, *i.e.* eradication of tick.

Protective inoculation may be performed by inoculating cattle with red-water gall-sickness blood before introduction into an infected country.

Theiler, however, recommended injection with 5 c.c. of blood containing pure *Anaplasma centrale.* The *centrale* infection produces a mild form of the disease and gives a high degree of immunity against *marginale.* Lignières recommends injection of anaplasm blood from sheep or goats through which the virus has been passed a number of times.

In Argentina Lignières failed to find *A. centrale* and found it too dangerous to inject *A. marginale* blood direct into cattle. J. T. E.

CONSTITUTIONAL DISEASES

SO-CALLED CONSTITUTIONAL DISEASES

UNDER this heading will be considered various affections whose etiology is at present obscure, and which used formerly to be regarded as *constitutional* diseases, that is, affections due to some innate peculiarity of the individual which was responsible for certain constitutional effects. Some of them are probably bacterial in origin, others probably due to disturbance of metabolism, while others are associated with derangement of functions with or without appreciable alteration of tissue.

RHEUMATISM

Rheumatics; "The Screws"

Rheumatism is a condition which affects the fibrous tissues of the body, principally those in the region of joints—the synovial membranes, ligaments, and the ends of bones—and also muscles, tendons, and tendon sheaths. It is always associated with a variable degree of pain, thus interfering considerably with general movement and locomotion.

Three forms of rheumatism are commonly recognized, viz.:

Acute articular rheumatism, or rheumatic fever.
Muscular rheumatism.
Chronic rheumatism.

It is doubtful whether there is any real difference between these forms beyond the regions affected and the severity of the attacks. Moreover, in the lower animals it is quite common to meet with articular and muscular rheumatism occurring simultaneously. It has been suggested that articular rheumatism is due to some infective agent, which, however, has not been satisfactorily demonstrated, and that muscular rheumatism is due to cold and exposure. It is more than probable, however, that both forms are due to some infective agent located in some part of the body as the alimentary tract or elsewhere, and secreting toxins which on absorption produce the fluctuating symptoms generally grouped under the term Rheumatism. It is also probable that more than one organism is capable of setting up the condition. It would therefore not be unreasonable to regard rheumatism as being a collection of symptoms of one or more affections, frequently of obscure origin. This view is supported by the frequency with which symptoms of rheumatism are developed in such diverse conditions as colitis, pyorrhœa, and even in influenza, and of similar symptoms due to the presence of the products of muscle fatigue after severe exertion. It may be difficult, however, to reconcile that view with the well-recognized predisposing causes to rheumatism which undoubtedly exist.

Predisposing Causes.—Amongst the influencing factors of rheumatism may be included climate, such as damp, cold, humid surroundings, and even lack of sunlight; exposure to cold as in the case of the east wind in the British Isles, and sudden changes of temperature. Certain foods also predispose to rheumatism, particularly those with a tendency to cause constipation. The existence of other affections such as colitis, pyorrhœa, cystitis, and metritis predispose to rheumatism. Age may also be a predisposing factor, young animals being more frequently affected with the acute forms, articular and muscular, while adults are more often affected with the chronic form of rheumatism. There is a widespread belief in a hereditary predisposition to rheumatism, but its existence is by no means proved. A rheumatic diathesis is also sometimes spoken of to account for the peculiar susceptibility of an individual to the development of symptoms of rheumatism. A previous attack of rheumatism frequently predisposes to subsequent attacks.

Rheumatism occasionally assumes the form of an outbreak, particularly in dogs and young pigs. In the former case it is frequently known as *kennel lameness*. The writer has also met with outbreaks of acute muscular rheumatism in sporting dogs where the affected animals were intensely infested with lice and fleas, and the circumstances pointed directly to a close association of these parasites with the causation of the symptoms.

Anatomical Changes.—In acute articular rheumatism changes may be observed affecting all the tissues of the joint. The synovial membrane and fringes are thickened and the periarticular tissues are swollen and infiltrated with yellow gelatinous liquid; the synovial liquid is increased, is yellowish-red and sometimes turbid, and may contain flakes of fibrin. The articular cartilage becomes softened and frequently eroded, leaving the bone exposed—sometimes the cartilage is replaced by fibrous tissue. When the spinal joints are affected the intervertebral cartilages may become ossified, leading to ankylosis of the spine. The bones themselves often show no change. At other times, however, there is a formation of new bone tissue along the edges of the articular cartilages, resulting in exostoses which may involve the ligaments and other tissues, and may ultimately lead to ankylosis of the joints. This is not infrequently met with in the stifle joints and fetlock joints of horses, and in the carpus and tarsus in cattle and dogs. The ligaments and tendons round the joint become thickened and sometimes elongated, and the infiltration usually extends into the tendon sheaths.

In muscular rheumatism frequently no changes are observable to the naked eye, but microscopically the fibres may be found to be in a state of " cloudy swelling " or even granular disintegration. In chronic cases there is an increase in the inter-muscular connective tissue. Cardiac lesions may also be met with following acute rheumatism, principally those of endocarditis affecting the valves; at other times pericarditis is present. The valves of the left side of the heart are most frequently involved, verrucose endocarditis being produced.

Symptoms.—In **articular rheumatism** or **rheumatic fever** the symptoms are usually rapidly developed with the production of acute pain in the joints and tendons, and with great lameness and inability to move. When standing, affected joints are usually somewhat flexed. If several limbs are affected simultaneously the animal may be unable to stand. The temperature is very high and may be anything from 106° to 108° F., while in less acute cases the elevation of temperature is only slight and subject to fluctuations. The pulse is hard, quick, and frequent (the so-called irritable pulse), respiration is blowing, and often abdominal. The joints most frequently affected in the horse are the stifle and fetlock joints. In other animals it is more frequently the hock and knee or wrist joints; any joints, however, may become involved. On manipulation the joint is found to be swollen, hot, and painful, and the patient objects to manipulation. The swelling and pain extend to the tendon sheaths above and below the joint. During the acute stage in a number of cases endocarditis is set up and may be responsible for fainting fits and sometimes intermittence of the pulse, but the chief cardiac irregularities are more likely to be observed in chronic rheumatism. During the acute stages the patient usually refuses food. There is a tendency to constipation, and the urine is reduced in quantity and darker in colour than normal. Rumination ceases in cattle and sheep, and the milk secretion is reduced in animals in active lactation. The duration of the attack is very variable. The symptoms may suddenly abate after a few days, while at other times they may last for weeks or even months. Recurrence is frequent, perhaps involving the same joints or other joints, and leads to considerable loss of condition. In horses, when the stifle joint is affected, the limb is suspended from the ground in the forward direction in quite a characteristic position. When the hocks are involved in cattle the animals frequently lie down and refuse to rise. If they are raised to their feet by means of slings they refuse to put any weight on them, and give the impression of being completely paralysed. The writer has known of instances where spinal injury has been consequently diagnosed and slaughter recommended, and during the interval pending the arrival of the butcher the animals have actually risen voluntarily, apparently owing to the sudden abatement of the pain in the joints.

Muscular Rheumatism.—In muscular rheumatism the symptoms are also rapidly developed. The affected muscles become tense and quivering when touched, and manipulation causes great pain. In the case of dogs they yell when touched or picked up, and they will sometimes cry out when moving without even being touched. The muscles most frequently affected are those of the shoulder and neck. When the shoulder is affected in the larger animals the fore limbs cannot be properly raised, a short step is taken, and the straw bedding becomes entangled round the animal's feet. A similar condition may also occur when the hind limbs are involved. When the neck is affected the head is usually extended or raised, and the patient resents any attempt at movement. The temperature is elevated several degrees, but rarely so much as in articular rheumatism. The pulse and respirations are markedly accelerated; the appetite is usually maintained, though the patient may be unable to feed owing to the difficulty of moving the neck for prehension. There is generally torpidity of the bowels with a tendency to constipation. When the fibrous tissues of the chest wall are affected, causing a painful condition of the side, the condition is called **pleurodynia.** If the fibrous tissues of the back are affected it is called **dorsodynia.** When the loins are affected it is called **lumbago,** and when the condition involves the regions of the croup and thigh the term **sciatica** is usually applied.

In **subacute** and **chronic rheumatism** the metastatic or moving character of the symptoms is more constantly observed. In this form the joints or muscles may be involved and the symptoms may occur suddenly as in the other forms. It may continue to affect the same area for some considerable time, or the symptoms may suddenly disappear to reappear elsewhere after a varying period of hours or days. It is in chronic articular rheumatism that the deformities of the joints are most likely to be met with. It is also in the chronic form that symptoms of verrucose endocarditis are most frequently developed.

Treatment.—The patient should be placed into an airy, well-ventilated, and well-drained house with equable temperature. In the acute form affected joints and muscles should be stimulated by means of liniments containing ammonia, turpentine, camphor, belladonna, and oil of winter green. A useful combination consists of lin. camph. ammon. ℥ss., lin. belladonn. and lin. sapon. pts. aeq. ad ℥viii., or antiphlogistine may be applied. The affected joints should then be bandaged with cotton wool, but no attempt should be made to reduce swollen areas by pressure. In the chronic affection of joints a mild blister of biniodide of mercury may be applied. Internally a smart purgative should first be given, and for this purpose calomel and sulphate of soda will be found equally useful. The bowels should subsequently be kept in a relaxed condition, either by repeated small doses of these agents or by the regular administration of paraffinum liquidum. The administration of salicylates, as salicylate of soda or of aceto-salicylic acid, will afford relief fairly rapidly in the majority of cases, and by many is regarded as an absolute specific. Salol may also be used, and is preferred by some practitioners. They may be combined with colchicum and also with bicarbonates of sodium or potassium. The combination of potassium iodide and quinine will frequently be found serviceable when salicylates have failed. In cardiac complications tincture of digitalis, strophanthus, squills, or nux vomica may be added.

Dosage.—See tables, pp. 544-546.

Where the condition is not too painful, light exercise is beneficial, preventing muscular wasting and reducing rigidity of joints ; general massage may also be of service.

The diet in rheumatism should be nourishing, concentrated, easily digestible, and laxative in character. In young pigs affected with rheumatism it is probably wise to consider the advisability of slaughter if they are in reasonably good condition.

Owing to the frequent association of lesions with rheumatism in other parts of the body a thorough examination of the patient should be made to ascertain if any other organs are involved or if any suppurative process can be discovered ; if so, proper treatment must be adopted. Thus the teeth should be examined for pyorrhœa ; the uterus and bladder should be examined, and the condition of the bowel and colon in particular observed ; while if the patient is infested with lice or fleas the parasites must be removed.

HÆMOGLOBINURIA IN HORSES

Hæmoglobinæmia ; Azoturia ; Black-Water ; Black Strangury

Azoturia is a condition peculiar to the horse tribe, and although it is frequently met with, its true etiology is still unknown. It always occurs under somewhat similar conditions, in which the horse, previously in hard-working condition, has been rested for a few days, during which time he has received full working diet. On being taken out to work the condition is produced with hardening of certain muscles, loss of muscular power, and the production of coffee-coloured urine. Horses kept under similar conditions of rest and feeding for longer periods than a few days apparently do not develop the condition. Both sexes are equally susceptible, and it occurs equally amongst heavy and light breeds of horses.

As already stated, the immediate cause of the condition is not definitely known. It used to be thought to be due to excess of urea in the blood and in the urine, but this has been proved not to be the case. It has also been considered by a number of authorities to be due to autointoxication, the toxin being either intestinal or muscular in origin. The theory put forward by M‘Fadyean appears to be the most feasible. He asserts that when a horse is in hard-working condition red blood corpuscles are produced in large numbers and destroyed during work, thus maintaining a balance. If the same horse is compelled to rest and at the same time receives his full working diet, then the corpuscles are produced in excess of the number broken down ; thus their number is largely increased during the rest. If the horse is then put to work in this condition before a compensatory reduction in production has been established, there is a sudden disintegration of the excessive numbers of corpuscles with the liberation of free hæmoglobin. The destruction of the corpuscles takes place chiefly in the capillaries of the muscles, which become choked, and by interference with local circulation degeneration of the muscle fibres of that part occurs. The muscle fibres then disintegrate and liberate more hæmoglobin. There are thus two sources of hæmoglobin, viz. the excessive red corpuscles and the broken-down muscle fibres. The liberated hæmoglobin is then eliminated by the kidneys, since it is greatly in excess of the amount capable of being dealt with as under normal circumstances by the liver.

Anatomical Changes.—The principal changes in azoturia in the horse, as revealed by post-mortem examination, are found in the blood and in the muscles. It has also been said that nephritis is produced, but whenever that occurs it is a secondary condition. The urine also shows important changes. It is very dark in colour and may be coffee-coloured. The colour is due mainly to hæmoglobin, and also to a derivative of hæmoglobin in a finely granular form. The urine is highly albuminous, but as a rule does not contain blood.

With regard to the blood itself, there is apparently no diminution of red blood corpuscles below the normal, but there is free in the blood plasma a quantity of hæmoglobin which is responsible for a red-tinted serum if the blood is allowed to clot. Crystals of hæmoglobin may be found after death external to the capillaries in the tissues, and they may also be found in considerable quantities in the form of casts in the tubules of the kidneys.

The muscular lesions are mainly met with in the sub-lumbar muscles, gluteals, muscles of the thigh, and sometimes the pectoral muscles, and in bad cases they are very easily detected. The principal macroscopic change is the alteration of the colour, which, instead of being more or less deep red, becomes partially decolorized and light pink. There may also be small hæmorrhages into the surrounding areas of the muscles. In mild non-fatal cases the degenerated muscle fibres may become removed by leucocytes, and the muscle may eventually become restored to its normal volume. In severe non-fatal cases a permanent breach or depression in the muscle may result.

Symptoms.—Occasionally hæmoglobinuria in the horse may develop when the animal is in the stable. In the vast majority of cases, however, there is the history already referred to of a short enforced rest of two or three days during which the horse has had his full working diet. Shortly after being taken out from the stable some alteration of gait is observed, such as a rolling motion or stiffness of the limbs, which may rapidly develop into acute lameness, usually in the hind limbs and occasionally of one or both fore limbs. The horse breaks out into profuse perspiration, and the muscles may be noticed quivering. If the horse is quickly pulled up and quietly returned to the stable, preferably in an ambulance, the symptoms may disappear without becoming more serious. If, however, he is compelled to proceed farther, he may fall to the ground. He generally attempts to rise, but finding his inability to do so he becomes violent and struggles. Cutaneous sensation is usually retained, and is shown by response to a pin-prick. If the muscles of his loins and quarters are then manipulated, they may be felt to be abnormally tense and firmly contracted, giving the impression as though swollen. They may at this stage be somewhat hotter than normal, but later the local temperature becomes reduced. In mild cases there may be no alteration of internal temperature, but in severe cases, particularly if there has been much struggling, the temperature may be elevated two or three degrees. The respiration is blowing and accelerated, while the pulse is frequent and hard. The mucous membranes are yellowish in colour due to hæmoglobinæmia. If urine is passed at this time or is drawn off with a catheter it is almost invariably found to be of a very dark colour, almost black, resembling stout or coffee. It is highly albuminous, and the colour is due to hæmoglobin and a granular derivative of blood pigment excreted by the kidneys. In very mild cases, however, discoloration may be very slight, the colour depending upon the severity of the attack. The reaction is usually alkaline. The quantity of urine may be increased in the early stages, but in bad cases the amount may be diminished as the result of choking of the renal tubules, and this may lead to uræmia with fatal termination.

Course and Prognosis.—In very benign cases the patient recovers fairly quickly. In those cases where paralysis at the onset is very slight, recovery may take place within a week. In severe cases death may occur in a few days, while a similar fatal result in cases with paralysis is common in two or three weeks. Death in the rapid cases is probably due to uræmic poisoning, while in the more prolonged cases death is generally brought on as the result of hypostatic congestion of the lungs and may be contributed to by the development of bed-sores and infection. The mortality averages from 40 to 50 per cent of cases. In favourable cases there may be no untoward sequels, while in other instances where recovery occurs there may be marked atrophy of the affected muscles coming on a week or so after the attack, and it may take months for such muscles to regain their normal volume. Occasionally there may be left subcutaneous breaches of the muscles, which, however, may cause no appreciable lameness with reasonable work unless they are very extensive.

Treatment. — *Preventive measures* should always be adopted in the case of horses in good condition being compelled to submit to a few days' rest. Such measures should consist in reducing the diet, particularly the corn, and substituting a bran mash, to which may be added a mild laxative and diuretic, such as sulphate of magnesia and potassium nitrate. The horse should also have daily walking exercise, even of short duration.

Curative Treatment.—As soon as an attack is noticed the horse should be conveyed in an ambulance to the stable, and this should be practised whether the horse is able to walk or not. On arriving at the stable, if the animal is able to support himself, he should be placed in slings, but not otherwise. In that case he should be provided with a deep bedding of straw in a roomy loose-box.

If urine is retained the catheter should be passed. The rectum should be emptied and a copious hot enema administered. In mild cases all that may be necessary medicinally is to administer a mild aperient, such as aloes or calomel. In severe cases a more rapidly acting purgative should be given, such as arecolin or eserine gr. i.-ii. This may be followed by a diffusible stimulant and mild diuretic, such as ammonium carbonate and potassium nitrate āā ʒii.-iv. and pulv. nux vomica ʒi.-ii. If there is much evidence of pain, chloral hydrate ℥i. may be administered. Various other agents have been recommended, such as adrenalin (ʒi.-ii. of 1-1000 solution), caffeine, digitalin, strychnine, and also the intravenous injection of perchloride of mercury. In the case of a horse lying down he should be massaged several times daily and turned over to reduce the danger of sloughs, while the parts which have been underneath should be sponged and rubbed with rectified spirits and water, 1-6 or 8. The food should be somewhat relaxing and not too highly nitrogenous—bran mash, green foods, steamed hay, carrots. For muscle atrophy, which may develop later, a mild blister may be applied.

Hæmoglobinuria in cattle is most commonly due to piroplasmosis, as in the case of common red-water. See pp. 231-236.

DIABETES

Two conditions are usually included under this term, viz.:

(1) Diabetes insipidus, or polyuria.
(2) Diabetes mellitus, or saccharine diabetes.

Diabetes Insipidus; Polyuria (Profuse Staling).—Polyuria is a condition in which excessive quantities of urine of low specific gravity are passed without there being any organic disease of the kidney. It is a symptom rather than a disease in itself. All species of animals are susceptible, but it is most frequently met with in the horse and in the dog. It is very frequently brought about by dietetic errors, such as mouldy hay, mowburnt hay, "doctored" oats, or by the accidental access to various drugs. Polyuria may also occur in the course of various specific diseases such as glanders and tuberculosis.

Symptoms.—The patient passes large quantities of urine which is usually lighter in colour than normal, and of low specific gravity owing to its containing a smaller proportion of solids. The actual amount of solids passed in a given time is usually greater than normal, but the proportion compared with the greatly increased amount of water is less than normal. It is associated with increased thirst and usually a well-maintained appetite. In mild cases and those of short duration there may be no other symptoms, but if the condition is continued it may give rise to considerable loss of flesh, a hard, harsh, staring coat, profuse sweating on light exercise, and inability to perform sustained work. The temperature is not altered. The bowels are somewhat irregular, with a general tendency to constipation.

The pathological condition of the kidney in this affection is simply one of active or arterial congestion, but it is not a serious condition unless it constitutes the early stage of nephritis or is symptomatic of some specific disease.

Treatment. — A mild purgative should be administered, preferably a saline, to remove offending matters from the alimentary tract, and also to afford direct relief to the kidneys. A complete change of diet and water-supply should be procured if either is open to suspicion. A course of iodides may be administered, such as iodide of iron or potassium, together with alimentary tonics.

Diabetes Mellitus; Glycosuria; Mellituria.—These are conditions in which there is an excess of grape sugar in the urine. The terms Glycosuria and Mellituria are usually applied to a temporary presence of sugar with or without excess of water, and the condition is not very serious in effect.

Diabetes Mellitus, on the other hand, is a much more serious condition, in which large quantities of sugar are continually passed, together with an increased amount of urine. The condition is not so common in the lower animals as in man, but it is not uncommon in the dog, and is less frequently met with in horses and other species. It appears to be more common in hot countries than in temperate zones; more common in females than males; and more frequent in old than in young animals.

The cause of diabetes mellitus is still uncertain, and several theories are put forward to account for it. It has been experimentally shown that injury of the floor of the 4th ventricle of the brain will result in a condition of sugar in the urine. It is probable that this operates by interfering with the functions of the liver. Saccharine diabetes may certainly arise from functional disease of the liver with disturbance of the glycogenetic function—the liver is either unable to arrest the sugar absorbed from the alimentary tract and to store it up in the form of glycogen, or the derangement permits of the reconversion of glycogen into sugar more copiously than is required. Some forms of diabetes are also said to arise from disease of the pancreas since experimental removal of the pancreas is followed by glycosuria. The writer has, however, met with an instance in a dog in which complete atrophy of the pancreas was unattended by glycosuria. Sugar is also present in the urine in certain well-defined affections such as epilepsy, asthma, and tetanus. A similar condition may also be produced by certain drugs such as morphia, chloroform, chloral hydrate, ether, and strychnine. The blood of animals affected with diabetes mellitus contains an excessive quantity of sugar.

Symptoms.—As already stated, this most frequently occurs in the dog, and is generally first indicated by loss of condition and progressive debility in spite of good appetite and generous feeding. There is marked thirst and some degree of constipation. The loss of flesh is very marked, and the abdomen becomes pendulous, while emesis frequently occurs. Cataracts are also not infrequent. There is an increased amount of urine discharged, lighter in colour than normal, but increased in specific gravity. On examination of the urine, large quantities of grape sugar will be found present, and may well exceed 2 per cent. The condition is progressive though usually slow, and may extend over several months, and in the majority of cases ultimately proves fatal.

In horses similar symptoms are observed, notably the increased thirst and appetite and the passage of excessive quantities of urine containing considerable quantities of grape sugar, which may be up to 4 per cent or 5 per cent of sugar. The loss of condition and flesh is very considerable, and death ultimately results from exhaustion in two or three months.

Diagnosis.—A positive diagnosis is made by the actual demonstration of sugar in the urine on analysis. Several tests are available.

(1) *Fehling's Test.*—Two solutions are necessary for this test. Solution A consists of copper sulphate in water, while Solution B is a mixture of sodium potassium tartrate and caustic potash in water. When required for use these two solutions are mixed and boiled. This is then mixed with an equal quantity of boiling urine, and if sugar is present a reddish-yellow precipitate of cuprous oxide will be produced without reboiling.

(2) *Nylander's Test.*—This is probably a better test than the preceding for the detection of small quantities of sugar. The reagent is made up as follows: 4 grms. Rochelle salt is dissolved in 100 c.c. of a warm 10 per cent caustic soda solution; 2 grms. bismuth subnitrate is added, and the mixture shaken. After cooling, the clear liquid is decanted from any undissolved matter. The test is carried out as follows: After boiling the urine and filtering to remove albumen, the filtrate is treated with $\frac{1}{10}$th its volume of the reagent and boiled for one to three minutes. If sugar is present bismuth is precipitated, giving a brownish appearance if in small quantity and black if copious. 0·1 per cent of sugar can be detected in this way.

(3) *Fermentation Test.*—This is probably the most certain method of detecting the presence of grape sugar. It is carried out as follows: A small quantity of yeast is added to a test-tube filled with the suspected urine. The mouth of the test tube is then closed with a cork through which passes one limb of a U-shaped tube. The test tube is then inverted and allowed to stand for twenty-four hours at warm room temperature. If sugar is present fermentation takes place, with the production of carbonic acid gas which rises to the top of the test tube and forces the urine downward into the U-tube. The production of carbonic acid gas under these conditions is positive evidence of the presence of grape sugar. It is well to prepare at the same time a control test, using a solution of grape sugar and yeast in order to make certain that the yeast is active.

Treatment.—The treatment of diabetes mellitus is not very hopeful and is mainly palliative. The diet must be carefully attended to, cutting down starchy foods as far as possible and substituting a diet rich in albuminoids and fats. This is very difficult in the case of herbivora, since their food obviously must contain considerable quantities of carbohydrates. Their diet, however, should consist mainly of good oats, linseed, and the best possible hay. Care should be taken that there is no suspicion of mouldiness or other defects of the oats and hay. For carnivora a meat diet should be prescribed, the best consisting of lean mutton and boiled paunches or tripe; an egg and milk diet is also excellent. Horse-flesh and liver should be avoided; the water-supply should be curtailed. Medicinally, drugs appear to have very little influence in glycosuria. Codein appears to be most effective, and following that opium or its derivatives, which reduce the glycogenetic functions of the liver. The salicylates may also be serviceable, and sometimes an improvement may be obtained by the use of quinine, potassium iodide, and arsenic. If there is any constipation it should be controlled by the administration of saline purgatives. There is a better prospect of recovery if the case has been taken in hand early, but as the condition is slowly progressive it is usually somewhat advanced before attention is drawn to it.

RICKETS

Rachitis

Rickets is a disease of young animals characterized by constitutional debility, together with enlargements of the ends of bones and a diminution in their resistance. Animals affected in the order named are: Dogs, pigs, sheep, foals, and calves. In lambs occasionally a considerable portion of a flock may be affected at the same time, while sometimes a whole litter of puppies or young pigs may be simultaneously affected.

Causes.—The etiology of rickets is still obscure, and has often been ascribed to deficiency of lime salts in the blood and in the food constituents. This is not substantiated experimentally, since it may occur in spite of an abundance of lime salts in the diet, while the experimental feeding of young animals with a diet deficient in lime salts has failed to produce the condition. In many cases it attacks young animals that are kept in too close confinement, especially in places that are badly lighted and ventilated, but against that it may be pointed out that it may and does occur amongst lambs kept out in the open air and getting plenty of exercise and good food. Defective quality of milk is also blamed for causing rickets, as may be brought about by improper feeding of the mothers. There is, however, no strong evidence in support of this view. In herbivora it has been attributed to excessive feeding of bran, and in pigs to feeding excessively on potatoes, while in dogs it has been variously ascribed to an excessive meat diet, and also to an absence of meat in the diet. More recently rickets has been ascribed to the absence of "vitamines" from the food. This theory is difficult to support in view of the fact that other animals receiving identical food do not become affected. (See illustration.) Heredity is said by some to influence the onset of rickets, while the presence of animal parasites in the alimentary tract has also been blamed for the condition. It is also possible that the affection may be due to some functional defect of the thyroid bodies. It has also been suggested that it is due to a micro-organism, but no such organism has been satisfactorily demonstrated.

It will thus be gathered that the etiology of rickets is still quite obscure, but whatever the origin may be the condition is evidently one in which the bone-forming cells are unable to abstract salts from the blood and to fix them in the proper manner to produce bone.

Anatomical Changes.—The ends of the long bones and ribs become enlarged and may develop protuberances. The shaft of the bone is often supple and becomes curved, and often thickened on the concave side; the periosteum is thickened, and between it and the bone tissue is a thick vascular layer. The bone tissue, even where it should be compact, is more or less spongy in texture and is easily cut with a knife. The cartilage between the shaft and the end of the bone is abnormally thick and often irregular. The flat bones, such as those of the head and the pelvis, may become very much thickened and bulging, and may easily be cut with a knife. Where the jaw-bones are affected, the nasal chambers may be obliterated and the upper jaw may protrude, while the teeth may become loose. When the lower jaw is affected, as sometimes happens in dogs, it becomes somewhat pendulous, and prehension and mastication are interfered with.

Symptoms.—The symptoms are absolutely confined to young animals and are usually developed slowly.

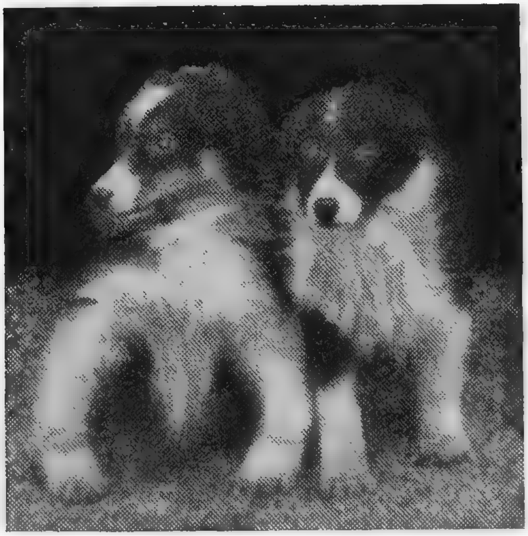


FIG. 63.—A puppy with rickets and a normal puppy: from the same litter and similarly fed. (Cousens.)

A certain degree of lameness may first be observed affecting one or more limbs, and on examination the ends of the bones, especially those of the limbs and the ribs, are found to be somewhat enlarged. They continue to increase in size, and may cause distortion of the joints. The shafts of the bones are soft and incapable of carrying the weight of the patient. The limbs consequently become much bent and deformed, and the animal lies considerably, with a disinclination to move about. Sometimes the hind limbs are affected to such an extent that they appear as though paralysed. When the head is affected the bones of the upper jaw are most frequently involved. They become swollen and may interfere with the proper closing of the mouth. At the same time the nasal chambers may become more or less obliterated, causing oral respiration. The upper jaw also protrudes beyond the lower. When the lower jaw is affected the rami become thick and softened, and may become partially pendulous. In these cases, which occur more often in puppies than in other animals, the prehension of food becomes very difficult or impossible, and owing to the inability to feed properly and as the result of general constitutional debility the patient loses flesh very rapidly. The bowels are irregular, sometimes constipated, other times diarrhœic, while the abdomen is frequently distended. The temperature is unaffected. The mucous membranes are generally pallid. The patients often develop symptoms of abnormal appetite, such as licking the walls and dirt, and the taking in of substances which they would ordinarily refuse.

The disease runs a chronic course lasting for months, but is more rapidly progressive in some cases than in others, and if untreated may terminate fatally from exhaustion or hypostatic congestion. On the other hand, if treated early a considerable number of cases recover, although permanent deformity of some of the limbs and joints may remain.

Prognosis of the condition is very difficult, since in spite of all measures some cases get rapidly worse, while others make a good recovery. In the case of any animals other than the horse, treatment is almost always advisable, but in the case of the horse, if the deformity of the limb is very great, it is probable that the animal even if he recovers will be unworkable. In such a case slaughter may be recommended.

Treatment. — *Preventive measures*, such as attention to hygienic conditions, the provision of reasonable exercise and proper diet are frequently recommended. These precautions undoubtedly tend to improve the vitality of young animals, but since the true etiology of the condition is unknown, their adoption as a preventive is somewhat speculative.

Curative Treatment.—It is as well to commence with a change of food and of pastures. Give the patient a generous well-balanced diet, and in young suckling animals the diet of the mother should be similarly considered. In the case of herbivora a generous diet of oats and good upland hay is undoubtedly the best, together with a little green food where obtainable. A lump of rock salt may be placed in the manger or feeding-troughs. In the case of dogs a generous meat diet should be given, together with cod-liver oil, which may be advantageously combined with liquid extract of malt and compound syrup of phosphates. In all species the administration of lime water and of blood tonics such as phosphates of calcium and iron, quinine, iodides and salicylates is advisable, and it is a good plan to add daily to the food a little green bone meal or bone marrow. Sheep's thyroids and thyroid extract are also claimed to have given good results. Attention should be given to the alimentary tract, administering digestives such as pepsin and hydrochloric acid, or alternatively tincture of nux vomica and bicarbonate of soda, with a view to improving digestion and assimilation. Very little local treatment is required. It may, however, be necessary to support limbs unable to bear weight by means of splints and bandages, but no attempt should be made to reduce swollen bones by pressure. The local application of stimulating liniments does not produce any better results.

OSTEOPOROSIS

Osteomalacia; Rarefying Osteitis; "Big Head"

Osteoporosis is a disease of bones confined to adults, and is developed after the bones have been once formed. It is a condition in which lime salts are removed from the bone, causing diminished resistance and allowing the increased formation of organic matter, with consequent enlargement. The disease may apparently affect all classes of animals, though in some countries it appears to be confined to equines, while in other countries serious outbreaks have occurred affecting considerable numbers of cattle and other species. Although the pathological condition of the affected bones in the different species is very similar, the clinical evidence points to the fact that osteoporosis in horses and cattle are probably independent diseases.

Etiology. — The same causes have been asserted for this affection as for rickets, and defects in the quantity or quality of food or of their relative constituents, as of lime and phosphates, have been suggested as responsible for the affection. This has especially been put forward to explain the occurrence of so-called outbreaks in seasons when the crops have been bad or where animals have been fed on food grown on poor, low-lying damp ground. As against this, however, is the fact that osteoporosis occasionally occurs in animals that are well fed and where no exception could be taken to the food. It is also suggested that the disease may be contagious, and outbreaks amongst cattle would sometimes appear to be due to some infective cause. Moreover, in horses successive cases have occurred in the stables after the introduction of an affected horse without any alteration of food or general management having been introduced. Against the view of contagion is the fact that when an affected animal is removed from the district to a locality where the disease is unknown, recovery frequently takes place with no tendency to spread to other horses. Another view as to its origin is that it is due to some defect of the thyroid gland, and certainly in some recorded cases in horses the thyroid gland was considerably enlarged. In the human being osteomalacia occurs most frequently in women during pregnancy, affecting especially the spinal bones and the pelvis. This, however, has not been particularly observed in horses though it has occasionally in cows. In horses in the British Isles osteoporosis is usually sporadic and at one time was most frequently observed in millers' horses, and was attributed to feeding excessively on bran (bran disease) and the husks of various kinds of corn. In fact, in millers' horses cases were often multiple. The disease is apparently of world-wide distribution.

Anatomical Changes.—Any bones of the body may be affected. It most frequently involves the head, the ribs, and the vertebræ. The limb bones may also be involved, and sometimes the pelvis. The affected bones become enlarged and swollen, often symmetrically; the bone substance is reduced in density and may become so soft as to be easily cut with a knife, while the periosteum is considerably thickened. At other times the bone may become brittle and result in spontaneous fractures. The attachments of the muscles and tendons may become loosened and the tendons become detached from their insertion. In the interior of the bones the spongy tissue may be replaced with a reddish gelatinous substance. In some cases there may be ulceration of the articular cartilage, which may actually extend to the bone substance. If the bone is macerated the ends or epiphyses become easily separated from the shafts. If the affected bone is boiled, removing the organic matter, a spongy network, the result of decalcification, remains, and is very fragile.

Symptoms.—In some cases the symptoms develop rather rapidly, but in the majority of cases the disease runs a more or less chronic course. It most often affects the head, which may be enlarged sometimes unilaterally, but much more frequently bilaterally, involving both the superior and inferior maxillary bones. The nasal chambers may become partially obliterated, interfering with respiration and causing roaring. The teeth sometimes become loose and displaced, thus interfering with mastication. When the limbs are affected there is loss of support with lameness and manifest instability, while the patient may have difficulty in rising. Fractures of the limb bones may be brought about, and at other times ligaments and muscular attachments may break down.

With regard to the general bodily condition, as the disease advances the patient loses flesh in spite of a well-maintained appetite and desire for food, even in those cases where mastication is not interfered with. Horses are unable to perform their ordinary work. When the vertebræ are affected there is irregular gait and difficulty in grazing. In osteoporosis the urine frequently contains an excessive amount of lime salts, in this respect differing from rickets.

In osteoporosis in cattle an abnormal appetite is frequently developed, as in pica (p. 277).

Prognosis.—It is practically impossible to forecast the termination of osteoporosis. If cases are taken in hand early, complete recovery occasionally occurs; partial recovery is more common, as the result of which horses may be rendered capable of light work. In the majority of cases, however, the condition is slowly progressive, and in spite of all efforts to check it the disease ultimately necessitates slaughter.

Treatment.—Although osteoporosis has not definitely been proved to be contagious, isolation should be practised where possible, and probably in the case of animals of small value slaughter is the most economic procedure. There should be a complete change of food and water, and a generous nitrogenous diet should be provided. The addition of molasses to the food is also strongly recommended. In the case of cows it is also suggested that they should not be completely milked, and that, if pregnant, they should be allowed to dry off at an early date. Castration of males and removal of the ovaries in females have been followed by beneficial results. Medicinally, treatment is on similar lines as that for rickets. A mild purgative should first be administered, to be followed by a course of blood tonics and general tonics, including phosphates, iron salts, sodium salicylate, potassium iodide, calcium carbonate, nux vomica, gentian, ground bone and bone marrow. The generous use of lime water may also be adopted. In those cases where enlarged thyroids are observed the administration of thyroid extract or sheep's thyroids may be tried. It may be necessary in some cases to support limb bones by means of splints and bandages, which should be well padded to avoid undue pressure. Disinfection of the buildings should be carried out and attention paid to general sanitation.

MILK FEVER

Dropping after Calving; Parturient Fever; Parturient Apoplexy; Parturient Intoxication; Vitulary Fever; Post-partum Paresis

Milk fever is a disease which occurs in milch cows and occasionally milch goats, and more rarely swine, at the time of parturition, and is characterized by nervous derangement frequently with partial or complete loss of consciousness and sometimes paresis. Of the synonyms quoted above those which suggest that the affection is a fever are inappropriate, since in the majority of cases there is no elevation of temperature, and in many instances there is a subnormal temperature.

The affection would seem to be a result of domestication and high development of milking propensities, since the condition rarely or never occurs in cows in the wild or semi-wild condition, and is met with mainly in those strains of milking cattle whose milk contains a large amount of butter fat, both actually and proportionately. It is therefore most likely to be met with in cows which yield very rich milk, or those which yield large quantities of milk not necessarily containing a high percentage of cream. The most susceptible breeds include the Channel Island cattle, milking strain of Shorthorns, Red Polls, Ayrshires and Holstein cattle. In these breeds milk fever is most likely to occur in those individuals that have been generously fed, especially during advanced pregnancy, and are consequently in high condition. It is more likely to occur after an easy than after a difficult parturition, though occasionally it may develop twenty-four hours or more previous to the parturient act. Milking the udder dry immediately after calving is thought by many to be a cause of milk fever. Obviously a newly-born calf never completely empties the mammary gland. This view of the cause is, however, unsatisfactory since it completely fails to account for those cases which occur previous to calving, where no milk has been removed from the udder. It mostly develops within three days after calving, but it may come on at any time within a week and possibly ten days after calving. The severity of the attack appears to be in direct proportion to the rapidity of its development after calving, and the longer the interval after the calving, the milder is the attack. It commonly occurs about the third calving, and one attack does not confer any immunity, but on the contrary would appear to predispose to attacks at subsequent calvings.

Etiology.—The actual cause of milk fever is not definitely known, though the conditions previously enumerated must be regarded as predisposing causes of milk fever. Many theories have been put forward to explain the origin of the affection, but most of them are purely theoretical and fail to carry any conviction. It has been variously ascribed to the following causes :

(1) *Cerebral Anœmia.*—This is thought to be due to stimulation of the vaso-dilators of the abdominal area, causing an increased flow of blood to those parts and producing anæmia of the brain. The removal of the fœtus is stated to still further increase the congestion of the abdominal vessels owing to the relief of pressure. A further contributing cause, and by some thought to be more important, is the great influx of blood to the udder for functional purposes at the time of parturition and immediately after.

(2) *Cerebral Hyperœmia.*—This was said to be caused by pressure of the abdominal wall and straining during parturition, producing engorgement of the vessels of the brain with effusions, and occasionally bringing about true apoplexy by causing rupture of some of the capillaries of the brain.

(3) *Fat Embolism.*—By this theory it was assumed that affected animals were the subjects of fatty degeneration, and that certain endothelial cells of vessel walls became detached and carried away by the blood-stream being arrested as emboli in the brain.

(4) *Leucomaines.*—By this theory it was suggested that a leucomaine was produced in some part of the body as the result of derangement of function of certain cells. Although no particular organ could be proved to be the origin it was thought that the udder was the most likely source of the leucomaine.

(5) *Intoxication.*—This is the view which is most widely held at the present time. This theory suggests that the symptoms are due to absorption of a toxin, the result of an infection of some part of the body. The organs suggested to be affected include the uterus, the alimentary tract, and also the udder. No toxins or affected material have been demonstrated in either of these situations capable of producing the symptoms. Moreover, as pointed out by Hutyra and Marek, in any cases where bacterial toxins and ptomaines produce paralysis and severe symptoms death almost invariably follows, while it is not uncommon for very severe attacks of milk fever to terminate in recovery.

(6) *Anaphylaxis.*—Hutyra and Marek suggest that milk fever is probably due to anaphylaxis, "brought about by a specific protein of the placenta or possibly the glandular cells of the udder, and that the absorption of considerable amounts of placentic or milk protein produces anaphylactic symptoms."

(7) *Mammary Neurasthenia.*—Wooldridge is of the opinion that the symptoms of milk fever are in all probability due to nervous causes originating in the nerve terminals of the udder. At the time of parturition these nerves are in a very high state of excitation preparatory to the assumption of the great functional activity of the gland. As the result of this, by reflex action the symptoms first of excitation followed by depression of the brain are produced. The higher the milking propensity of the individual, the greater will be the receptivity of the nerve terminals to the stimulation, and thus the greater susceptibility to the development of the symptoms. This theory is largely supported by

clinical experience of the results of treatment both by such agents as chloral hydrate and bromides, which reduce nervous excitability through their action on the higher nervous system, and also even to a greater degree by the excellent results which follow the distension of the udder either by air or oxygen, or any liquids. It would appear to the writer that the good results of the latter treatment are solely due to distension of the udder and pressure on the nerve terminals, reducing their sensitiveness and functional activity, since practically the same results are obtained, no matter what the distending agent may be, whether air, pure oxygen, or solutions of potassium iodide, or chinosol, or iodine, or salt, or even sterile distilled water.

Symptoms.—Symptoms may develop at varying intervals after parturition or they may have developed twenty-four hours or so before calving. The condition only develops after an easy parturition and usually only in cows calving indoors. On rare occasions, however, it may develop in cows allowed to calve out in the pastures. The first indication of anything wrong is a general uneasiness with an occasional moan, and a paddling or swaying movement of the hind limbs. At other times there is bellowing and occasionally muscular spasms, which, however, are of very short duration. The cow usually neglects her calf and walks with a staggering gait. The pupils are dilated and twitching of the iris may be noted. In cases that develop rapidly these symptoms may be overlooked and the cow may first be noticed to drop. She often attempts to rise, sometimes succeeding, but frequently in vain, and if still conscious she may knock herself about on finding her inability to rise. There is rarely, however, much violence, but when it does occur there is suspicion that the case is one of true apoplexy. The loss of consciousness (coma) comes on sometimes very rapidly, while in rare cases the cow may actually ruminate after lying down. As a general rule, however, the cow lies on her brisket and turns her head round, resting on the chest wall with her nose pointing towards the flank. Another position not so frequently adopted is with the head stretched out resting along the ground. At other times the cow may roll over on to her side, a position which is very conducive to tympanites, which may terminate fatally from asphyxia. At this time there may be stertor or snoring, which is often regarded as an unfavourable sign. The eye is dull and the corneal reflex is lost and the upper lid droops. There is a temporary cessation of defæcation and urination and also a marked reduction of secretion of milk, the udder being somewhat relaxed and limp. The respirations are usually deep and slow, while the heart is accelerated but beats somewhat feebly; the pulse is soft and small, sometimes almost imperceptible, and from 80-100 per minute. The body surface and the extremities are cold. The internal temperature usually commences by being elevated 1° or 2°, but rapidly declines and usually becomes subnormal—96°-97° F. Pyrexia seldom occurs, and when it does it may be attributed to complications such as a uterine infection. During the coma there is paralysis of the pharynx and œsophagus, with inability to swallow, and consequently saliva dribbles from the mouth and occasionally regurgitated food escapes.

Course.—In mild cases seen early and put under appropriate treatment the condition may pass away in a few hours, and it is remarkable how quickly, even in cows that have gone down, ability to rise is restored by appropriate treatment. In many cases cows apparently quite paralysed are able to regain their feet within half an hour. In other cases the affection lasts one or two or even three days, after which the cow may rise, but if coma lasts longer than three days recovery rarely occurs. The first sign of returning consciousness is the resumption of spontaneous defæcation and micturition. The cow then raises her head and looks around and may quickly rise to her feet. The appetite gradually returns and secretion of milk is resumed. The pupils become normal. The muscles in such cases appear to regain their power very quickly.

In other cases where apparent recovery from the milk fever occurs, paralysis of the hind limbs remains as a sequel. The cow appears constitutionally well so far as appetite and excretions are concerned, but is unable to support her weight with her hind limbs and is insensitive to a pin prick. Bed sores may then occur on the hips and the outside of the thighs, and slaughter may be rendered necessary. In other cases, two or three days after consciousness has returned and the animal apparently well on the way to recovery symptoms of pneumonia develop, due to saliva and other substances having passed down the trachea. This condition usually terminates fatally several days later.

Prognosis.—It is difficult to forecast definitely the termination of a case of milk fever during the early stages of the affection. By recent methods of treatment, however, in cases taken in hand early the mortality has become reduced to a very low figure and is probably not more than 10 per cent, while many practitioners are able to point to a sequence of recoveries with a still higher percentage. The death-rate, however, is very largely increased where drenching with medicines, etc., has been practised during the course of the affection. The severity of the attack and consequent mortality also appear to bear direct relationship to the rapidity with which symptoms are developed after parturition. Relapses may also occur after apparent recovery, but an early repetition of treatment in such cases is usually effective.

Differential Diagnosis.—Milk fever should be distinguished from a condition known as **adynamic fever,** from **true apoplexy,** and from **parturient metritis.**

Adynamic Fever (Loin-dropped).—This is a simple condition of debility after parturition in which the animal prefers to lie down rather than to stand. There is little or no disturbance in bowels, temperature, or respiration, and no loss of consciousness.

Apoplexy.—This is more likely to be confused than some other affections, and may produce symptoms somewhat similar to those of milk fever. In true apoplexy, however, coma is rarely so complete, but the comatose or semi-comatose condition is usually longer than in milk fever. There is also a greater tendency to a preliminary stage of violence, while sometimes one pupil may be found dilated while the other is contracted. In the later stages bed sores and paralysis of the hind quarters are more likely to occur than in milk fever.

Parturient Metritis.—This condition, which is due to entrance of organisms into the womb after parturition, rarely comes on until three or four days after calving, and is always associated with a rise of temperature and other febrile symptoms. There is also frequently retained fœtal membranes and an offensive uterine discharge.

Treatment of Milk Fever.—*Preventive measures* should be adopted in all likely subjects during the last few weeks of pregnancy. The diet should be reduced and not too highly nitrogenous, and daily exercise should be given in the case of cows kept indoors. It is also a good practice to allow suspicious cows to calve out at pasture. This step is recommended on account of the rarity of cases of milk fever occurring in cows while at pasture, though milk fever occurring under such conditions is not unknown. A mild laxative as of sulphate of soda or sulphate of magnesia should be given about fourteen days before parturition and repeated seven days later. Some Continental veterinarians recommend venesection with the extraction of about six pints of blood six or seven days before calving. After calving the milk should be drawn in small amounts from the udder. In many cases, however, in spite of all precautions, milk fever will develop.

Curative Measures.—Procure the best hygienic conditions possible with good ventilation, and provide plenty of bedding to diminish the danger of bed sores. If the cow is already down she should be propped on her brisket either with bundles of straw or sacks stuffed with straw to prevent tympanites. She should be turned over from one side to the other every three or four hours and the legs well massaged before and after turning; a little milk may be removed each time. During coma the rectum should be emptied manually, and the catheter should be passed two or three times a day. In cases of violence a cold swab or ice-pack should be applied to the poll. The best therapeutic measures consist in distending the udder at the earliest possible moment, which should be done if possible as soon as premonitory symptoms are noticed. Judging by the results it is not of great importance which of various agents are employed for distending the udder, each of them giving good results. Probably the best procedure consists of injecting first a quantity of chinosol solution 40 gr. dissolved in 1 qt. of previously boiled water, ¼ injected into each quarter of the udder. This should be followed by the injection of filtered air, or better still, oxygen from a cylinder. The udder should be firmly inflated and the teats closed either by tape or a clip, which should be left on for one or two hours. The mammary gland should then be well and thoroughly massaged in order to distribute the injected material throughout the tubules. In some cases distinct improvement may be seen in less than an hour, and in that time the cow may actually rise to her feet. If she fails to do so in five or six hours the treatment should be repeated. In the majority of cases the cow will be on her feet within twelve hours. It is highly important that every precaution should be taken to ensure asepsis both of instruments and materials employed. Neglect of this precaution is a fruitful source of mammitis. Teat syphons should be boiled and conveyed to the cow protected by sterile cotton wool or used direct from the sterilizer. If filtered air is to be employed the filtrix must be sterile. It is a good plan to pump the air

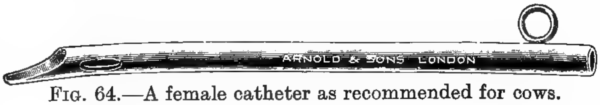


Fig. 64.—A female catheter as recommended for cows.

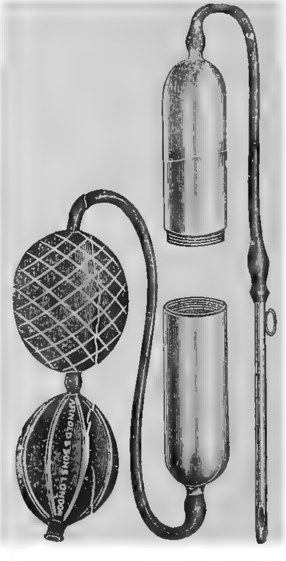


Fig. 65.—Apparatus for inflating the udder with filtered air.

through a disinfectant such as chinosol solution in a flask. The cotton wool filtrix often employed is frequently infected and a source of mammitis. Fresh material should certainly be used for every case. Several useful auxiliary measures may be adopted, such as the hypodermic injection of strychnine (liq. strych. ʒii.) or caffeine ʒi.-iss., adrenalin (1-1000) ʒi.-ii. In cases of violence if the cow still retains consciousness sedatives may be administered with advantage, such as potassium bromide and chloral hydrate āā ℥i., but it is unwise to resort to drenching under any pretext unless the cow is able to swallow freely, on account of the great danger of producing drenching pneumonia. As an additional precautionary measure the uterus should be mopped out and any retained fœtal membranes removed and an antiseptic pessary inserted.

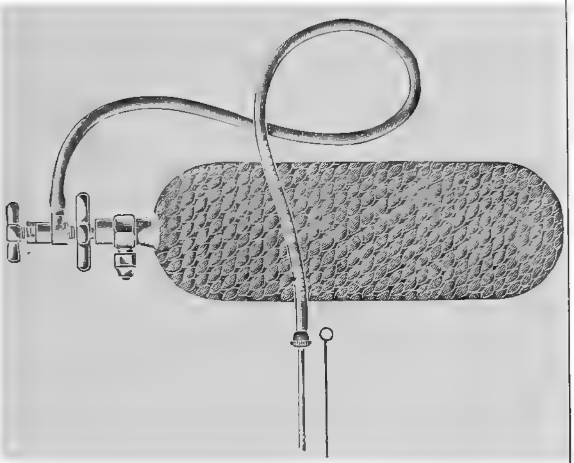


Fig. 66.—Oxygen cylinder and apparatus for inflating the udder.

Schmidt, to whom all credit is due for the introduction of the intra-mammary method of treatment, recommended the injection of 10 grm. of potassium iodide dissolved in a litre of sterile water and injecting $\frac{1}{4}$ into each quarter of the udder. This injection is, however, not infrequently followed by mammitis owing to the difficulty of maintaining asepsis. Moreover there appears to be a greater subsequent diminution of milk yield after the use of potassium iodide. Other solutions employed from time to time include iodine solution ($\frac{1}{2}$ per cent to 1 per cent), lysol solution ($\frac{1}{2}$ per cent to 1 per cent), and salt solution (gr. iv.-℥i.), but, as already stated, the writer prefers chinosol followed by air or oxygen, since it is the least likely to be followed by mammitis or reduced milk supply.

When the cow becomes convalescent the udder should not be stripped for ten or twelve hours, after which a small amount of milk can be drawn several times daily. The diet should still be restricted and gentle exercise permitted.

In cases where paralysis occurs as a sequel, injections of strychnine may be resorted to and a blister applied along the spine. Special attention in these protracted cases must be paid to massaging the hind limbs. In some cases the paralysis is undoubtedly the result of the anterior tibial nerves becoming involved with bruising, thus producing paralysis of the extensors of the feet. The writer has observed this injury to the nerve in a number of cases on post-mortem examination. G. H. W.

DISEASES OF THE THYROID GLAND

Very little is known of the functions of the thyroid. They are, however, very important, and their arrest gives rise to serious results. In fact, the thyroid gland may be regarded as an absolutely vital organ.

Diseases of the thyroid gland usually causes enlargement of that body. An enlarged thyroid is generally known as a *goitre.* On the other hand, atrophy of the thyroid with a reduction in size is also met with. In each case there may be interference with the functions of the thyroid, more particularly in atrophy, which may result in cretinism in the young, or in myxœdema in adults.

Atrophy of the Thyroid ; Thyroid Insufficiency ; Hypothyroidism ; Myxœdema ; Cretinism

In adults, atrophy of the thyroid, or its removal, is followed by myxœdema—general wasting with dryness of the skin surface and swelling of the subcutaneous tissues, impairment of the mental powers, and frequently with convulsions followed by paralysis. In goats, however, the thyroid may be removed with impunity (author's experience).

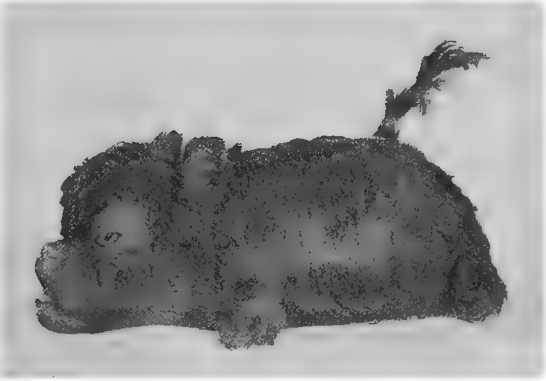


Fig. 67.—A bovine cretin.

In the developing fœtus and in the newly born, atrophy of the thyroid causes a condition known as **cretinism,** which is arrest of physical development. An example of this condition occurs frequently in Dexter cattle as the result of which monstrosities are produced. The head is usually larger than normal, and in shape somewhat resembles that of a bull-dog, while the legs are often only 2 inches or 3 inches long, and the tail arises well forward on the croup. Cretinism has also been observed in dogs, but is much less common.

Treatment.—Young animals born with this condition are usually destroyed, but unless stillborn, treatment may be adopted, since the grafting of a piece of thyroid or the daily administration by the mouth of raw sheep's thyroid or a glycerine extract of thyroid is likely to be followed with good results; at any rate calves may thus be reared till they become fit for slaughter for food. Myxœdema in adults may be successfully treated in the same way.

Goitre ; Bronchocele ; Struma

These terms are applied to any enlargement of the thyroid body, and may thus include four varieties of goitre, viz. the parenchymatous, cystic, malignant (adenoma or carcinoma), and the exophthalmic.

The causes of goitre are unknown. It has been ascribed to something in the water or herbage in certain districts where the disease may be prevalent. It has also been suggested that it is due to micro-organisms, and it is stated to have been experimentally transmitted to goats. In the lower animals it is most frequently met with in dogs and lambs and in foals, and occasionally it occurs in the form of outbreaks where all the puppies of a litter may be affected, or a considerable proportion of the lambs in a flock. Enlarged thyroids are frequently met with in adults, and unless very large they rarely cause any serious symptoms.

Symptoms.—There are rarely any constitutional symptoms in adults, but in young the thyroids may rapidly increase in size and cause death from asphyxia by pressure on the trachea and pharynx. In adults the enlargement of the thyroid body may be unilateral or bilateral, and may develop slowly or rapidly. In the horse it is commonly bilateral, and the swelling attains the size of a duck's egg close to the larynx. On manipulation the gland is easily moved under the skin, and occasionally it may be found to pit on pressure. Sometimes goitre causes pressure on the œsophagus and difficulty in swallowing. In dogs also the affection may be unilateral or bilateral, and the enlarged gland frequently gravitates to a position in the neck somewhat lower than normal. It is freely movable in this situation, and is thus distinguished from inflamed lymphatic glands which may be met with in the same region.

Treatment.—If the condition is unilateral, that portion of the gland may be excised. When cystic an incision may be made in the cavity, or it may be punctured and a weak solution of iodine injected, such as tincture of iodine ʒi., distilled water ʒii. If incised, the wound heals fairly rapidly with ordinary care. The external application of potassium iodide ointment or iodine ointment is also recommended, but is of doubtful benefit. The internal administration of tincture of iodine, potassium iodide, liquor arsenicalis, or a thyroid extract, is often followed by good results. In dogs the writer has frequently obtained complete recovery by giving thyroid extract m. v. every morning and potassium iodide gr. v. every evening, in each case before food. During the course of this treatment a generous diet must be provided, otherwise the patient very rapidly loses condition.

In the case of malignant tumours of the thyroid, excision is the only available treatment, and, to be successful, must be performed in the early stages before the disease has penetrated the capsule and begun to involve the surrounding part.

Exophthalmic Goitre ; Graves' Disease ; Basedow's Disease.—In this condition there is derangement of the functions of the thyroid, accompanied by an enlargement of that gland, prominence of the eyeballs, and nervous and vascular excitability. The cause of the condition is unknown, but the symptoms are thought to be due to increased activity on the part of the gland. The condition is therefore just the opposite to myxœdema.

Symptoms.—The symptoms usually develop slowly. The first thing observed is enlargement of the thyroids, one or both sides being involved, and of firmer consistence than normal. Following this, protrusion of the eyeballs may be observed, with staring eyes and often a squint, together with an overflow of tears. About this time cardiac irregularities occur such as palpitations, with shaking of the body at each heartbeat, and noticeable pulsation of the superficial arteries. The pulse is full and strong, but the temperature is not altered. Alimentary disturbances may also be met with—loss of appetite, diarrhœa, and vomiting in animals capable of emesis. There may also be change in general habits of the patient, which usually becomes more or less lethargic.

Treatment.—Treatment of exophthalmic goitre is not very hopeful in veterinary patients. Partial extirpation of the thyroid affords the best results. Medicinally, heart sedatives such as digitalis and strophanthus may be found useful. Parathyroid extract and pituitrin have also been used and good results claimed. Perfect rest and quietness should be provided.

G. H. W

LOCAL DISEASES

DISEASES OF THE DIGESTIVE TRACT

Stomatitis; Inflammation of the Mouth

All inflammatory affections of the mouth or the buccal mucous membrane are included in the term stomatitis. Stomatitis may be diffuse or localised. It may be a simple or superficial affection without any serious import; while, on the other hand, it may be deep-seated and serious in its effect. Even in the most superficial and benign forms stomatitis is likely to cause inappetence, principally through interference with prehension, and the difficulty or indisposition to take up food. Fortunately the buccal mucous membrane is endowed with great powers of spontaneous healing. Inflammation of the mouth may also occur in the course of certain specific or contagious diseases, such as "foot-and-mouth" disease and the various forms of variola (pox).

Stomatitis in the Horse

Diffuse or Catarrhal Stomatitis.—This condition is very frequently met with, and generally involves a more or less extensive area of the mucous membrane of the mouth. It may be caused by the administration of drugs insufficiently diluted, such as ammonia, turpentine, lysol, chloral-hydrate, alcohol (neat spirits). It may also be due to accidental access to irritating substances. Blistering oil administered by mistake for linseed oil has been responsible for it, and so also has the inadvertent licking of blister ointment from various parts of the body. These forms of diffuse stomatitis may also be termed *chemical* stomatitis, and are often associated with inflammation of the pharynx.

Symptoms.—Frequently the first thing that is observed is indisposition of the animal to feed, together with dribbling of saliva, often frothy, from the mouth. The patient usually objects to examination of the mouth. The lips and tongue may be swollen, and the general mucous membrane a brighter or deeper red than normal. In severe cases of chemical stomatitis there may be shreds or flakes of the mucous membrane in the discharge, and the seats from which they have been exfoliated will be noticed eroded. The submaxillary and pharyngeal lymphatic glands may in severe cases be somewhat enlarged, and slightly hot and painful to manipulation. There is usually no systemic disturbance except in very severe cases, when the temperature may be elevated 2 or 3 degrees. The patient usually evinces a desire for food by whinnying at the approach of his attendant with food, but when it is placed before him he usually refuses it, unless it is of a soft, more or less succulent nature, or semi-solid as in the case of a mash. In those cases where the throat or pharynx is also involved, attempts at swallowing are likely to produce fits of coughing.

Treatment.—Nursing, with special attention to food, is of most importance. It is not imperative, however, that a horse should have any solid food for the first 24 or 36 hours, and in fact it is often best that such foods should be entirely withheld for that period. This frequently allows healing to commence, and to be fairly well advanced before any solid food is given, which would otherwise somewhat retard the healing process. On the other hand, gruel or milk or hay tea may be offered, and the horse will usually drink these fairly readily. On the second day a bran mash or boiled oats may be given, and following that, cut grass or other green foods. Feeding with dried foods and with long hay should not be resumed until healing of the buccal mucous membrane is complete. For a period, corn should be crushed and damped, hay should be given either steamed or in the form of damped chaff. Medicinally, relief may be afforded by the administration of mild antiseptics and sedatives in the form of electuary. The best combination consists of boracic acid, chlorate of potash, and extract of belladonna, made up to the required consistency with powdered glycyrrhiza and treacle. Mouth washes may also be useful, such as boracic acid and alum in 1 to 2 per cent solution. Other useful agents are hydrogen peroxide, permanganate of potash, carbolic acid $\frac{1}{2}$ per cent.

Vesicular Stomatitis.—This form is frequently called *aphtha*. It consists of the development on the mucous membrane in any situation of vesicles or blebs. A vesicle is an upheaval of the epithelium from the surface of the subjacent structures, forming a blister-like elevation, and containing a thin watery or serous liquid. This form of stomatitis is not common in horses, but it is occasionally met with. Its etiology is not well understood. It is undoubtedly in some instances due to micro-organisms, and therefore contagious. In other instances it is due to irritants which may have been taken in with the foods.

Symptoms.—The condition is rarely a serious one, and causes very little inconvenience to the patient. Several animals may be affected simultaneously. There may, however, be some inappetence and dribbling, and sometimes a slight elevation of temperature. On examining the interior of the mouth, the blister-like elevations may be seen, greyish-white in colour, often surrounded by a red or hyperæmic zone. They may be observed in any part of the mouth, but are most frequently seen inside the lips and cheeks, and below or on the sides of the tongue. If one of the blisters be punctured, it will be seen to contain a more or less clear liquid, which, however, in advanced stages may become turbid, but rarely pus-like. They are usually small in size and regular in outline, but if they are very numerous they may coalesce, forming comparatively large lesions. Such cases are rather more serious, and may also be associated with slight swelling of the sub-maxillary or pharyngeal lymphatic glands.

Treatment of Vesicular Stomatitis.—As the condition is usually somewhat benign it does not call for any special measures beyond those which have already been recommended for catarrhal stomatitis. The application of mild astringents and antiseptics assists the healing process. Attention should be paid to the food, which should be soft or demulcent in character. Isolation should be practised to reduce possible contagion.

Ulcerative Stomatitis.—This form is sometimes met with in horses, and consists in the formation of ulcers in various parts of the buccal mucous membrane. The ulcers are often preceded by vesicles. Ulceration is also frequently due to slight injuries or wounds which may have been produced by irregularities of the teeth. Thus they may occur on the inside of the cheeks owing to sharp outer edges of the upper molar teeth, or they may occur on the side of the tongue from a similar condition of the inner edges of the lower molars.

Symptoms.—One of the most pronounced symptoms is the dropping of masses of food from the mouth during mastication (quidding), associated with a dribbling of stringy saliva, offensive in odour, and sometimes blood-stained. On examining the mouth, the horse frequently offers some resistance, and food material may be seen collected between the teeth and the cheeks. The shape of the ulcers varies but is usually somewhat irregular. The breath is frequently offensive, owing to septic infection of the ulcers, the edges of which may be found necrotic or gangrenous (yellowish-green in colour). Ulcers may also be discovered on the inner aspects of the lips, and are then usually secondary to vesicular stomatitis.

Treatment.—If the condition is due to irregular teeth, the latter must be attended to, and rasping of the sharp edges is frequently sufficient. All loose food material should be removed, and ulcers may then be dressed with single application of tincture of iodine after any necrotic edges have been removed. Further treatment is on similar lines to that recommended for catarrhal and vesicular stomatitis.

Mycotic Stomatitis.—This condition, which is sometimes called *thrush*, is only rarely met with in horses. Cases have, however, been recorded due to various yeasts and moulds. When present it affects the tongue, and other parts of the mouth, and may extend into the pharynx. The lesions are first slight vesicular elevations followed by ulceration.

Treatment is comparatively simple, but special attention should be directed against the possibility of contagion. Feeding and watering utensils should be kept for the individual animal, and should be constantly disinfected. Medicinally, similar measures may be taken as in the case of ulcerative stomatitis.

Contagious Pustular Stomatitis.—(See Contagious Diseases, p. 65).

Stomatitis in Cattle

The buccal mucous membrane of ruminants is much less sensitive to irritants in general than that of the horse. Nevertheless cattle and sheep are not infrequently affected with various forms of inflammation of the mouth, some of which are contagious, and others non-contagious. One therefore meets with catarrhal and vesicular forms of stomatitis as in horses, due to similar causes, and giving rise to similar symptoms.

Treatment.—Perhaps the most important feature is the feeding. It must be remembered that cattle are unable to gather their food properly with an inflamed tongue, and consequently they should be brought up from the pastures and fed with cut grass and other succulent or soft foods.

Ulcerative Stomatitis in Cattle.—This form in cattle is rarely due to injuries from the molar teeth as in the case of horses. It is, however, comparatively common, occurring in the course of actinomycosis, to the article on which the reader is referred.

Desquamative Stomatitis.—A condition of the mouth of cattle occurs in certain parts of Ireland, which has been described under the above name. It is also known locally as "dirty mouth." Its main importance is due to the fact that it has been confused with foot-and-mouth disease, although there are marked differences between the two conditions. It principally affects cattle on pasture, and although the cases may be multiple there is some doubt as to its method of spread. It appears, however, to be infective, and animals probably contract it during the prehension of foods contaminated by the discharges of affected animals. The period of incubation appears to be less than a week. According to Craig, it seems to affect only young animals under two years old.

Symptoms.—The condition appears to be purely local, affecting the base of the tongue, the gums, and dental pad. The lips are only rarely involved. The mucous membrane of these parts becomes markedly thickened owing to the accumulation of considerable quantities of epithelium in more or less circumscribed areas about half an inch in diameter. These may extend until almost the entire tongue is involved. Patches of the epithelium may become detached, leaving depressions. The condition can be readily distinguished from foot-and-mouth disease by the fact that there is no formation of vesicles, and by the colour, which is brownish, whereas in foot-and-mouth disease it is usually white. There is no evidence of contagion to other species, and none of the pigs or sheep on the premises become affected, and, moreover, there are no foot lesions. The condition passes through its course in from two to three weeks, but appears to cause no serious inconvenience to the patient. Indeed its presence is frequently unsuspected until the mouth is examined for other reasons.

Treatment.—When the condition is observed the accumulated scales should be scraped off and the parts dressed with weak antiseptic solutions. It is well to separate the affected from the healthy to reduce the danger of infection.

Necrotic Stomatitis in Calves.—This affection is due to the bacillus of necrosis (*bacillus necrophorus*) and is a contagious disease. It is principally met with in young calves, and is serious and frequently fatal. The lesions affect principally the tongue, sides of the cheek, and the gums, and consist of the appearance of yellow necrotic patches. The condition is dealt with under Contagious Diseases, to which the reader is referred (p. 81).

Stomatitis in the Sheep

Catarrhal stomatitis may be met with in the sheep, as in other animals, and due to similar causes. It is, however, a comparatively rare occurrence, and does not call for special consideration.

Vesicular Stomatitis in the Sheep.—This condition is not infrequently met with, and consists in the production of blebs or vesicles about the lips and tongue. It occurs most frequently in the autumn when sheep are feeding on stubble. It is said to be contagious, and various organisms have been described as associated with it. That point is, however, doubtful. It is more probable that the condition is mechanically produced by injury with the hard stubble.

Symptoms.—The sheep are unable to feed properly, and consequently lose condition fairly rapidly. Frequent movements of the lips indicate the presence of some irritation. On examining the mouth small vesicles, which are usually pointed, may be seen inside the lips.

Treatment is comparatively simple, and consists in removing the sheep from the stubble, and providing them for a few days with soft food and cut grass. Antiseptic and astringent mouth-washes may be used, as in diffuse stomatitis.

Ulcerative Stomatitis.—This condition occurs in ewes and lambs running together, and is sometimes called *malignant aphtha* of the sheep. It is frequently associated with a gangrenous mammitis in the ewes, and is undoubtedly a contagious affection. The mouth lesions are much more common in the lambs than in the ewes, and consist of the formation of ulcers and scabs, which affect the lips and may extend inside the mouth and outside the lips to the face. It is a very serious affection, and is fatal in a considerable number of cases largely owing to the inability of the lambs to feed. In the ewes the mammitis is much the more serious condition. The condition is described fully under Contagious Diseases.

Papillomatous Stomatitis.—The lips of sheep sometimes become affected with small wart-like growths, which, however, rarely invade the cavity of the mouth. The chief trouble is caused by the interference with prehension of food, and consequently affected sheep are likely to fall off considerably in condition. The origin of the condition is not properly understood. It is thought, however, by many to be due to feeding on stubble. There is a natural tendency to spontaneous recovery, but the careful application of a mild caustic, such as silver nitrate, or tincture of iodine, may accelerate the disappearance of the growth. Affected sheep must also be removed from the stubble and fed with soft foods.

Stomatitis in the Dog

Catarrhal Stomatitis.—This condition is not very frequently met with, owing to the fact that the dog rapidly expels any irritant from the mouth. Occasionally chemical stomatitis is produced, which results in dribbling of saliva and indisposition to feed, or to lap liquids. In such cases recovery is usually fairly rapid, and treatment is quite simple, consisting in the application of weak mouth-washes of boracic acid and alum. If the inflammation persists the dog must be fed artificially, though he is likely to object if the condition remains painful.

Ulcerative Stomatitis in the Dog.—This is a condition in which there develop eruptions on the buccal mucous membrane and lips, and which sometimes spread up the sides of the face. It is sometimes called *canker of the mouth*. It is undoubtedly infective, as is demonstrated by the fact that if one lip is affected the corresponding part of the other lip meeting it also becomes affected. The condition is rather serious, and may have fatal results. The ulcers are comparatively superficial, and there is frequently a superficial portion of dead tissue, yellow in colour, attached to the surface. They are most commonly seen inside the lips opposite the tushes, and inside the cheek extending backwards from the angles of the mouth. Their presence, although serious, is frequently overlooked. The breath of affected animals is extremely offensive. Systemic symptoms are set up consisting of general depression, indisposition to feed, loss of condition, emaciation, and progressive debility. The temperature is frequently elevated 3 or 4 degrees, and the pulse is weak and frequent. Dogs of all ages appear to be susceptible, but it is most frequently met with in dogs past the prime of life.

Treatment.—The patient should be isolated and kept in a warm well-ventilated place. The ulcers should be exposed, and any yellow necrotic tissue must be snipped off. They must then be cauterized somewhat severely with either tincture of iodine or pure carbolic acid if not too numerous. In the latter case care must be taken that the phenol does not run over other parts of the mouth, and, moreover, one should be careful with its use owing to the great susceptibility of dogs to the effect of carbolic acid. Any bad teeth or tartar on the teeth must be removed. Subsequent treatment consists in mopping out the mouth with antiseptics, such as permanganate of potash solution or hydrogen peroxide or alum. This should be done frequently throughout the day. The lesions must be examined daily, and any fresh necrotic tissue observed should be similarly removed. Constitutional treatment is of equal importance to the local treatment, and consists in administration of blood and general tonics such as compound syrup of phosphates, cod liver oil, and extract of malt. Quinine is also useful. The strength of the patient must be maintained by generous feeding with meat extracts or milk with white of egg, Benger's food, lactol, etc., which must be given in small quantities and frequently.

Gangrenous Stomatitis in the Dog.—This condition is frequently met with in dogs associated with gastro-enteritis and the condition sometimes called Stuttgart dog disease. The mouth lesions in many respects resemble those produced by phosphorus poisoning, the teeth become coated with brown, blood-stained, and have a very offensive deposit. The gums swell up, become soft and pulpy and necrotic, and the teeth may become loose and fall out. The tip of the tongue at first becomes livid, and then yellow and necrotic. This may extend in some cases to the whole of the free portion of the tongue, which may slough off. The animal refuses all food, and very rapidly loses condition and becomes hidebound. There is great thirst for water. The condition, as already stated, is frequently associated with gastritis, and vomiting is a frequent feature, particularly after drinking water. *Treatment* to the mouth consists in the removal of all the necrotic tissue by means of the scissors and curetting the part, which must then be severely cauterized with pure tincture of iodine. Loose teeth must be removed, and the mouth must be frequently swabbed with mouth washes, either of permanganate of potash or boracic acid or salicylic acid or hydrogen peroxide. This condition, with its more serious gastric and intestinal complications, is dealt with more fully under Contagious Diseases, p. 182.

Papillomatous Stomatitis in the Dog.—This is a form of contagious warty growths of the mouth which may be met with in other species as well as the dog, but is by far the most common in the last named. The lesions consist of small wartlike growths covered with a dense epithelium, and white in colour. They may be few in number, or they may be very numerous. They are principally met with on the lips and inside the cheeks, and may extend backwards involving the pharynx. In some cases the visible mucous membrane is almost entirely covered with them. Their infective nature is indicated by the fact that if one lip is primarily affected the other lip may also become affected at the point where it comes in contact with the growth. There have also been what might be described as outbreaks of the affection occurring amongst a number of dogs kept together, either members of the same litter or packs of hounds. The condition is most frequently met with in puppies or young dogs, and is extremely rare in adults.

Symptoms.—In the majority of cases there are no appreciable systemic symptoms, and the presence of the growths may not even be suspected until the mouth is examined for other reasons. The growths are then seen to be more or less numerous, and to have dense white covering of epithelium. They are quite painless on manipulation. There are no constitutional symptoms unless prehension is interfered with, or the growths are so situated on the cheek as to come between the molar teeth. In such cases, owing to the difficulty of feeding, the affected dog loses condition, and in very exceptional cases may actually become emaciated. Occasionally similar growths may be found on the skin, and these are principally cases of auto-inoculation from the mouth.

Treatment.—In the majority of cases there is a natural tendency for the growths to disappear spontaneously. Their disappearance, however, may be accelerated by the internal administration of potassium iodide in daily doses of gr. ii.-gr. v., and the local application of tincture of iodine. Methylene blue has also been recommended for the condition, either administered in gr. ii. to gr. v. doses, or by local application. In the writer's hands, however, this agent has not met with encouraging results.

If the growths interfere with taking up or masticating food, then liquid foods and artificial feeding must be resorted to.

Chemical Stomatitis.—Mouth lesions are sometimes produced as the result of poisoning by chemical agents which may have been taken accidentally or administered maliciously, or may have been used over too long a period for medicinal purposes. Irritant drugs like ammonia and turpentine, if insufficiently diluted, cause diffuse catarrhal stomatitis. Other examples are furnished in poisoning by phosphorus, lead, and mercury.

In phosphorus poisoning, the mouth lesions consist of a yellow discoloration with necrosis of the buccal mucous membrane and the tip of the tongue. Phosphorus poisoning is generally due to the dog or cat having eaten of phosphorus vermin-killer such as rat paste (see "Phosphorus Poisoning," p. 512).

In lead poisoning, the effect in chronic cases is to cause a deposition of lead under the gums which results in the production of a bluish-grey discoloration. This may be brought about by animals licking paint, or in some particularly susceptible animals, such as toy dogs, being kept in a newly painted building. In the latter cases, even the very minute amount of lead in the atmosphere which is responsible for the odour is actually sufficient to cause lead poisoning (see "Lead Poisoning," p. 510).

In the chronic mercurial poisoning the gums may also become affected with a bluish discoloration, and there is a formation of ulcers towards the margin of the gums, which also swell up, and teeth may actually become loose. There is also an offensive odour from the mouth. This condition may follow the absorption of mercurial poisons from the skin (see "Mercurial Poisoning").

Stomatitis in the Cat

The mucous membrane of the mouth of the cat may become inflamed similarly to that of the dog, and thus we may meet with catarrhal and ulcerative stomatitis, producing symptoms exactly corresponding to those in that animal, and calling for similar treatment.

Vesicular Stomatitis in the Cat.—A peculiar and somewhat severe form of vesicular stomatitis occurs in cats and is undoubtedly of a contagious nature. The chief lesions are met with round the free margin of the tongue, which becomes reddened. On observing closely one may also find there numerous small vesicles containing clear liquid.

Symptoms.—The cat is usually noticed to be very ill, becoming listless and refusing all food, liquid or solid. There is a considerable quantity of stringy saliva hanging from the mouth. On examining the mouth, the presence of vesicles may be observed round the edge of the tongue, both at its tip and along the sides. Breathing is somewhat hurried, and the temperature is elevated to 104° F. or 105° F. Constipation is also usually present.

Treatment.—The condition is readily amenable to treatment, but, if neglected, is likely to prove fatal, principally from lack of food and debility. It is well first to administer a purgative, such as calomel gr. i. The mouth should be swabbed out several times daily with boracic mouth-wash, and a mild febrifuge such as acid aceto-salicylic gr. ss. to gr. i. given twice a day. Artificial feeding must be resorted to, and it will usually be found that the cat will readily swallow milk placed in the mouth by means of a spoon; in fact it would appear that the inappetence is mainly due to the soreness of the tongue, and not to any real loss of appetite. The administration of the purgative and the artificial feeding together would appear to be the most important parts of the treatment.

PHARYNGITIS

The pharynx is subject to almost all the diseased conditions that may be met with in connection with the mouth, of which it may almost be regarded as an extension.

Catarral Pharyngitis in the Horse.—This condition is very common in the horse, and is perhaps most frequently met with in the course of certain specific catarrhal diseases such as strangles and influenza. It may, however, also be due to the same cause as catarrhal inflammation of the mouth.

Symptoms.—The first thing usually observed is partial loss of appetite and some difficulty in swallowing (dysphagia). Attempts to swallow may set up fits of coughing. The throat may be swollen both inwardly and outwardly, and manipulation of the throat sets up coughing and gulping, and may be objected to by the patient. The sub-maxillary and pharyngeal lymphatic glands are usually somewhat swollen, hot, and tender, the degree depending upon the severity of the affection. There is also usually a catarrhal nasal discharge, which at first may be thin and watery but at length becomes thicker. If there is much swelling of the throat, food materials may be returned through the nostrils. This is particularly observed when drinking. Similarly the swelling may also interfere with respiration, and cause roaring during both inspiration and expiration together with exaggerated flank movements. The head is usually extended, and the patient presents a somewhat dejected appearance. In the horse, catarrhal pharyngitis is frequently accompanied by some fever, the temperature rising to 104° F. or 105° F.

Croupous or Diphtheritic Pharyngitis in the Horse.—This condition is rarely met with, but when it does occur it is a rather severe condition and causes considerable distress. There is a pseudo-membranous deposit on the mucous membrane of the throat, and shreds or flakes of these may be included in the nasal discharge, which is also frequently offensive. In other respects the symptoms produced correspond with those of severe catarrhal pharyngitis.

Œdematous Pharyngitis or Phlegmonous Pharyngitis.—In this condition the deeper layers of the pharynx are involved in the inflammatory process, the mucous membrane becoming swollen and often ulcerated. As a result of this, there is great narrowing of the fauces, with considerable obstruction to swallowing, and severe dyspnœa. In some cases it comes on very suddenly, and may disappear almost as suddenly, while in other cases, if not speedily relieved, death may result from asphyxia.

Œdema of the pharynx may occur in horses as the result of poisoning by *Lathyrus sativa* (see "Lathyrism").

Treatment of Pharyngitis in the Horse.—Owing to the tender condition of the throat and the difficulty in swallowing, the food must be soft or liquid in character and demulcent. Cut green foods or scalded hay chaff may be given, or mashes of boiled linseed or bran or boiled oats. Oatmeal gruel, milk, and hay tea are also useful. If any hay is given it should be previously steamed. It is often advisable for the first 24 or 48 hours to completely withhold all solids, leaving the patient nothing but tepid water or milk. Horses do not suffer in any way from this temporary fast, and the withholding of the solids may permit the pharynx to undergo resolution more rapidly.

Medicinally, inhalations may be resorted to with steam, and such medications as eucalyptus, creosote, terebene, or phenol. These undoubtedly assist the expulsion of the discharge from the nose and pharynx. *Internally*, local antiseptics and astringents may be given in the form of electuary, as in the case of stomatitis. Probably one of the best combinations consists of boracic acid, chlorate of potash, flowers of camphor, extract of belladonna, glycyrrhiza and treacle. Sulphate of magnesia ℥ii.-℥iv. or iodide of potassium ʒi.-ʒii. may be given in the drinking water. Chlorodyne ʒi. in glycerine ℥i. affords relief, administered twice or thrice daily, being tipped into the mouth so as to be swallowed slowly. *Externally*, relief may be afforded by the application of hot fomentations, by poultices, or by the application of a counter-irritant, such as camphor liniment, turpentine liniment, a weak mustard plaster, or a mild cantharides blister. One should also be prepared at any time to insert a temporary tracheotomy tube if the dyspnœa becomes severe. This is particularly the case in the œdematous form, as met with in lathyrism.

Pharyngitis in cattle is a comparatively rare occurrence. It is occasionally met with as an extension of inflammation from the mouth, and, as such, calls for no additional treatment beyond external application to the throat, such as a mustard plaster or a mild blister. The pharynx in cattle, however, may become involved in the course of certain specific diseases such as actinomycosis, rinderpest, and malignant catarrh, to the articles on which the reader is referred.

Pharyngitis in the Dog.—This is a fairly common condition, and may occur in the course of certain specific diseases such as distemper and Stuttgart disease. It may also occur as a primary affection. Catarrhal pharyngitis is the commonest form in the dog.

Symptoms.—The first thing usually observed is a hacking dry cough, which may last for a few days and is accentuated by feeding. It frequently gives the impression to the owner of a foreign body, such as a bone, being stuck in the throat. After coughing in this way a little frothy saliva may be expelled from the mouth, and is often mistaken by the owner for vomiting. Vomition, however, does occasionally occur in uncomplicated pharyngitis. After a few days the cough becomes more insistent and moist in character, and is accompanied by a catarrhal nasal discharge. On examining the mouth, and depressing the tongue, the pharynx can be seen red and somewhat swollen, and frequently covered with frothy mucus. On manipulating the outside of the throat it is found to be somewhat tender and sometimes swollen. Such manipulation produces gulping or sometimes a movement and sound comparable with an effort to remove such a thing as a fish bone from the throat. Systemic symptoms may be shown in the form of elevation of temperature in the more severe cases, and in those which are secondary to specific affections. In mild cases recovery may take place spontaneously if the dog is kept in comparatively warm surroundings.

Treatment of Pharyngitis in the Dog.—The *external* application of stimulants to the throat, such as *linimentum saponis* or *linimentum terebinthinæ,* will frequently afford considerable relief, as also will applications of hot stupes (flannel wrung out in very hot water and sprinkled with turpentine) or thermogene wool. *Internally,* local sedatives and antiseptics may be given as in the case of stomatitis. Considerable relief is also afforded by the administration of a mixture consisting of equal parts of compound tincture of camphor and glycerine, with or without a little water added. If there is any fever, febrifuges should be given, such as aceto-salicylic acid or sodium salicylate gr. ss. to gr. v., or quinine sulphate gr. i. to gr. ii. Medicated inhalations will afford some relief, and also facilitate removal of the nasal discharge.

With regard to food, only those foods of a demulcent nature should be offered, such as milk with white of egg, or barley water, finely minced meat, or shredded fish carefully freed from bones. In severe cases, however, such solids are likely to be refused, probably with advantage to the patient.

Abscess of the Pharynx or Post-pharyngeal Abscess.—This is a surgical condition which is most frequently encountered in horses and cattle. In the former it may occur as a complication in the course of strangles, while in cattle it is most frequently due to actinomycosis. In either species it may also result from the inoculation of small wounds of the pharynx. Post-pharyngeal abscess may occur in the wall of the pharynx, or it may involve the pharyngeal lymphatic glands.

Closely allied to this condition is the occasional occurrence of tumours of the pharynx, which in some cases are pedunculated (polypus).

Symptoms are those of pharyngitis accompanied with roaring and dyspnœa, intermittent often if due to polypus. This may be very intense, and if not relieved may actually lead to asphyxia. In favourable cases the abscess may burst into the pharynx, and the exudate is either expelled from the mouth or, more frequently, swallowed. Sometimes it escapes into the larynx, and may set up bronchitis or broncho-pneumonia. In other cases the abscess may burst outwardly, and when this occurs it is the more favourable outlet.

Treatment of pharyngeal abscess is mainly surgical. If the pharynx be examined a fluctuating point can generally be discovered in the swollen tissues, and this is the point at which relief must be afforded. The abscess must be opened and drained if possible from the outside of the throat. If the fluctuation is only to be met with internally, then a mouth-gag or speculum should be inserted, and the abscess punctured inwardly. These are cases in which discretion must be exercised as to the best time for puncture. The operation should not be performed before the abscess is mature for fear of injury to the large vessels in the region. On the other hand, it should not be left too late, and should be performed as soon as the fluctuating point is clearly discoverable. The abscess cavity should be irrigated with a suitable disinfectant, such as iodine solution 1 per cent or 2 per cent, carbolic acid 2½ per cent, or perchloride of mercury 1-2000. If a polypus is present it must be removed by snaring with a wire or a fine chain ecraseur. It may be necessary to relieve the dyspnœa by the insertion of a temporary tracheotomy tube. Medicinally the same treatment should be applied as in the case of simple pharyngitis, except in those cases of actinomycosis in cattle, when the best results are obtained by the administration of potassium iodide (see "Actinomycosis").

Paralysis of the Pharynx.—This is a comparatively rare condition, but it may be met with in any species of animal. The cause is not well understood, but it is probably due to interference with the nerve supply of the pharynx either locally through the peripheral nerves, or centrally through some injury to brain centres governing the pharynx.

Symptoms.—The condition may come on suddenly, or may appear gradually. The affected animal is unable to swallow, or can only swallow with great difficulty. Liquids may be returned by the nostrils. The difficulty of swallowing sets up coughing. The patient generally makes an effort to feed, but is quite unable to do so, and although he may dip his nose into a pail of water, he is quite unable to drink any of it. Loss of condition rapidly follows through starvation.

Treatment is far from satisfactory; in a few cases spontaneous recovery may occur. Nerve stimulants, such as strychnine, may be injected hypodermically, and blisters applied to the throat, but such methods are usually disappointing in results. Food may be administered by means of a stomach pump or in the form of nutrient enemata. Recovery is not very hopeful, but it may occur if the animal's strength can be maintained long enough by artificial feeding.

Blaine or Ting.—This is a peculiar condition of cattle which is said to occur in the western counties of England, attacking more commonly highly fed animals, and generally when being driven to or from the market.

Symptoms.—These are said to come on quite suddenly. The animal becomes rapidly breathless and affected with intense dyspnœa, lies down, and threatens to asphyxiate if not speedily relieved. On examining the mouth a large bleb is said to be found, involving the posterior part of the dorsum of the tongue and part of the pharynx. It may attain the size of an egg. The drover generally treats the condition by means of his pocket-knife, with which, on opening the mouth, he incises the swelling. After a short while the breathing is relieved, and the animal rises and can proceed.

There is some doubt as to whether this is a genuine condition or not. It is probable that some of the cases of so-called blaine are due to the animal being overdriven. The tongue then becomes drawn back towards the pharynx, and the mucous membrane of the dorsum is thrown into a fold which is mistaken for a bleb or large vesicle. This certainly would give rise to acute dyspnœa, and when the mouth is opened and the tongue drawn well forward the condition is immediately relieved without necessitating the use of the knife. In other cases the condition is probably one of urticaria affecting the tongue and pharynx.

Urticaria in cattle occurs sometimes, affecting the tongue and pharynx in the form of suddenly-appearing circumscribed swellings which are elastic in consistence but do not contain any free liquid. Similar swellings occur on the outside of the cheek in the region of the throat. It is a benign condition and does not call for drastic measures. Relief is usually afforded by the administration of a mild purgative together with a laxative diet.

Pus in the Guttural Pouches.—This condition is due to an extension of catarrh from the pharynx up the Eustachian tube into the guttural pouches. It gives rise to symptoms of exaggerated pharyngitis with increased dyspnœa owing to the swelling. The condition is usually unilateral, but may also be bilateral, and causes a swelling behind the angle of the jaw, and under the parotid gland. It gives rise to an intermittent nasal discharge which is thick and creamy, and is particularly observed when the head is depressed or when the swelling is manipulated externally. This may be one form of so-called nasal gleet. In chronic cases of long standing the pus may become inspissated and converted into small bodies called chondroids. These are usually brown in colour, and they take on a fairly high polish owing to constant friction with one another. In some rare cases the condition becomes complicated by the introduction of gas-forming organisms, resulting in a tympanitic condition of the sac with very marked swelling externally, which is resonant on percussion.

Treatment.—Relief of this condition is purely surgical. In recent cases pus may be drawn off by the passage of a special cannula up the Eustachian tube into the pouch. The pus, however, is usually rather thick and does not flow down the cannula very freely. Better results are obtained by opening the pouch externally, great care being taken to avoid injury to large vessels and nerves in the immediate neighbourhood (see "Hyovertebrotomy"). In the tympanitic condition relief can be given by the use of the trocar and cannula. It is not, however, very satisfactory, as the tympany constantly recurs.

Pharyngeal Choking (see p. 1004).

DISEASES OF THE ŒSOPHAGUS

The œsophagus is not subject to many diseased conditions which give rise to clinical symptoms. It is apparently not a very sensitive structure, and is, moreover, endowed with a fairly thick mucous membrane. Most of the affections are only discovered on post-mortem examination.

Spasm of the Œsophagus; Œsophagismus.—This condition is occasionally met with in animals, particularly the horse. It is, however, comparatively rare, and its cause is not well understood. It would seem, however, to be a nervous condition, and is probably brought about reflexly as the result of indigestion. It may also occur in some well-defined conditions, such as œsophageal choking, or in dilatation following stricture of the œsophagus.

Symptoms.—These may be developed very suddenly, and usually while the animal is feeding. He stops feeding and appears to be very distressed, gulping, and then drawing the chin in towards the chest, followed by retching somewhat resembling attempts to vomit. This may be accompanied by the discharge of saliva and food materials by the mouth or nostrils, or both. It may also be associated with a peculiar crowing sound produced at the larynx. On examining the gullet down the left side of the neck it may be felt to be firm and contracted. The lips may also be seen to be spasmodically moved, and general uneasiness is apparent.

Treatment.—In some instances the condition will pass off just as quickly as it arose, and except for the temporary slight distress does not call for any immediate treatment. In prolonged spasm, however, relief may be given by the application of hot fomentations down the neck, and also by the injection hypodermically of small doses of atropine or morphia, or chloral hydrate may be administered per rectum. After-treatment consists of the administration of a laxative and attention to the diet, which should be somewhat restricted for a while, and of a succulent nature. As indigestion may be the original cause of the trouble, a course of tonics should be given, such as arsenic and nux vomica.

Paralysis of the Œsophagus.—This affection may be met with, but is by no means a frequent occurrence. The cause is not well known, but is probably due to some lesion of the central nervous system. It may also occur in the course of certain specific diseases, such as rabies.

Symptoms.—The condition may be associated with paralysis of the pharynx, in which case little or no food is passed into the gullet. If the pharynx is not affected, food may accumulate or become arrested in the upper part of the œsophagus. It may be felt in this position just behind the throat, and practically amounts to a condition of partial choking. There is not, however, so much obstruction to the swallowing of liquids.

Treatment.—This is by no means satisfactory. Any obstruction in the œsophagus must be removed, preferably by manipulation in the upward direction from the outside of the neck. It is not advisable to force the arrested food material down the œsophagus into the stomach if it can be avoided, owing to the distance it would have to be pushed (see Treatment of Choking, p. 1006). Attempts to restore the muscular activity of the œsophagus may be made by local application of blisters to the neck, and by the internal administration of nerve stimulants, such as strychnine and arsenic. The animal's strength must be maintained by means of thin liquid foods, such as milk, oatmeal gruel, barley water, hay tea, etc. Prognosis of the condition is, however, unsatisfactory.

Inflammation of the Œsophagus; Œsophagitis

Inflammation of the œsophagus is only rarely met with. It may be caused by powerful irritants, such as insufficiently diluted medicines, as, for instance, ammonia, acids, or alkalies. It may also be traumatic, due to foreign bodies, such as nails, etc., in the forage, in horses and cattle, and sharp bones or meat skewers in dogs and cats. Œsophagitis may also be present in some specific affections, such as foot-and-mouth disease, rinderpest, etc. Except in the case of the foreign bodies, the condition is accompanied by inflammation of the mouth and pharynx.

Symptoms.—These are only observed in the more severe cases; the milder cases are likely to pass unnoticed. The principal symptom is in connection with swallowing, which is either difficult or causes some discomfort; the head is usually extended, there is gulping or frequent attempts to swallow. In deep-seated cases there may be regurgitation up the œsophagus, and a mucous or blood-stained discharge may be expelled through the mouth or from the nose. In dogs and cats, when due to various foreign bodies, vomiting frequently occurs, and the animal shows great distress, and has an anxious expression on the face. In these cases perforation of the gullet is likely to occur, and the serious nature depends to some extent on the situation of the arrested foreign body. If it is in the upper or cervical portion of the gullet the foreign body may pierce the skin and even escape without very serious consequences; if in the base of the neck between the first pair of ribs, or if in the intra-thoracic region, perforation is likely to be followed by a putrid or gangrenous pleurisy which is fatal. In other cases the perforation occurs about the region of the diaphragm, and the foreign body, if a skewer, may pass out between the last ribs. It is remarkable that in a large number of these latter cases complete recovery follows.

Treatment.—In cases other than those due to foreign bodies relief may be afforded by the administration of narcotics or anodynes hypodermically, such as morphia, or anodynes by the mouth in the form of electuary, as in the case of pharyngitis. Mucilaginous foods, such as milk, linseed tea, oatmeal gruel, may be given, and solids or dry foods should be withheld for several days. When the condition is due to a foreign body, surgical interference is necessary (œsophagotomy).

Dilatation of the Œsophagus.—This is a condition in which the lumen of the œsophagus becomes more or less permanently enlarged, either uniformly (*ectasia œsophagi*) or in which the wall is dilated in a circumscribed manner at the particular part of the wall (*diverticulum œsophagi*). The former is generally due to gradual pressure of accumulating food materials, resulting from a constriction lower down the gullet. The shape of the swelling is more or less fusiform, and only rarely sacculated. The wall usually consists of the whole of the structures of the œsophagus, which have been stretched by the pressure. Diverticula are generally produced as the result of a rupture of the muscular coat of the gullet, the mucous membrane being pressed into the breach by food substance. They are usually, therefore, bi-sacculated.

Symptoms.—Dilatations of the gullet are not easily recognised in the early stages. They may be slowly developed and not observed until they become rather extensive. The first indication is frequently that, during a meal, an animal suddenly stops feeding without any apparent reason, and shortly afterwards spasm of the œsophagus takes place, expelling some of the food material through the nose and mouth. If the dilatation is in the region of the neck, the swelling may be easily discoverable on the left side of the jugular furrow, and on manipulation it is doughy in consistence and somewhat fluctuating. If left alone it has a tendency to gradually get smaller, and ultimately disappear only to recur at subsequent feeding. In those cases where the dilatation is intrathoracic the case is frequently diagnosed as one of true vomition, especially as it is accompanied by the convulsions of the inferior cervical muscles. On examining the material that is expelled, however, it will be seen to consist of a mixture of frothy saliva and food which may be decomposed, but which shows no signs of undergoing digestion. As the condition is likely to be more or less recurrent the affected animal loses considerably in condition, but yet may be able to work satisfactorily for years before fatal consequences ensue. In other cases rupture may occur from over-distension, while death may also be brought about by suffocation or from pneumonia arising as a complication.

Treatment.—Curative treatment is not likely to be effective, and one must therefore adopt palliative measures, which chiefly consist of attention to diet. During an attack, relief is best afforded by simply placing a bucket of water on the floor in the stable, and allowing the animal to drink whenever he wishes. The swelling may be manipulated in the endeavour to facilitate the passing of the food materials down the œsophagus. Obviously, this can only be performed when the dilatation occurs in the region of the neck. All food material should be withheld until the condition is relieved, which, under these conditions, will occur spontaneously after a longer or a shorter time. As a routine measure, the food given to such animals should constantly be soft or sloppy, all dry or long foods being withheld. The subject should be fed at frequent intervals in small amounts. By such means the animal can be kept in a workable and useful condition for years.

Stricture of the Œsophagus; Stenosis of the Œsophagus.—This is a condition in which the lumen of the gullet becomes reduced. It may be due to an actual constriction of the œsophageal wall following upon some injury, or it may be due to external pressure, as in the case of tumours or abscesses, in the region of the neck or in the chest.

Symptoms.—Stenosis of the œsophagus as such is rarely or never diagnosed. An affected animal will commence to feed quite normally, and after swallowing a few times, if the food is at all firm or dry, he stops feeding. Repeated attempts at swallowing occur, and occasional attempts to feed, but the food is likely to be returned by the nostril. The taking of liquids is not seriously impeded unless the stenosis is very marked or an obstruction has occurred in front of it with solid ingesta. If that were so, the water would be returned through the nostrils. When this condition occurs in cattle the normal eructation of gases (belching) is prevented, and consequently tympanites or "blown" supervenes. Stricture or stenosis of the œsophagus is usually followed by dilatation in front of the constriction.

Treatment.—This is not very successful in so far as permanent results are concerned. The same principles must be observed as in the case of dilatation, special attention being paid to frequency of feeding, with soft or sloppy foods. Where tympany occurs in cattle, relief must be afforded by puncturing the rumen with the trocar and cannula. If the ruminant is in otherwise good condition, one should consider the advisability of slaughter for food.

Papilloma of the Œsophagus.—The mucous membrane of the gullet is occasionally the subject of papillomatous or wartlike growths, similar to those occurring in the mouth, particularly in the dog and sheep. They are usually small, though they may be multiple, and consequently may not give rise to any symptoms. If, on the other hand, they are large or very numerous, they may set up obstruction and dysphagia. On rare occasions a single large papilloma has been met with causing an obstruction, and owing to repeated traction through the swallowing of food has caused an intussusception of the gullet.

Treatment.—This is rarely called for in the cases of multiple small papillomata owing to the difficulty or impossibility of diagnosis. In the case of the single large growth the definite nature of the condition beyond some obstruction is not recognized. Œsophagotomy may be performed, and the growth, when its true nature is discovered, excised with successful results.

DISEASES OF THE STOMACH IN RUMINANTS

The first three compartments of the stomach of ruminants (rumen, reticulum, and omasum) are not concerned with the immediate process of digestion, and may with advantage be regarded as dilatations of the œsophagus. Their functions are mainly concerned with the mixing and softening of food, and permitting various fermentative changes preparatory to the process of true digestion, which will subsequently take place in the fourth compartment (abomasum, or true digestive stomach) and intestines.

Diseases of the Rumen

Impaction of the Rumen; Plenalvia; Engorgement of the Rumen.—This is a condition of over-distension of the rumen with solid food. The causes of impaction are very varied, and may depend upon the quantity and quality of the food, or on certain defects of the walls of the stomach itself. The kinds of food which are most likely to give rise to the condition consist of dry innutritious substances, such as may be obtained on pastures towards the end of a dry summer or in the early autumn, also rye grasses, straw, etc. It may also result from greedy feeding, especially with such agents as bad hay, which otherwise the animals might refuse, and with which they have been tempted by sprinkling with such things as treacle, etc. Impaction may also arise from muscular weakness of the rumen walls (atony) or from deficient secretions. It is also conduced to by lack of water and insufficient exercise, and also by a sudden change from succulent to dry feeding.

The condition known as "clover-blown," which is due to an animal engorging itself with fresh succulent clover, is a combination of impaction and tympanites.

Symptoms.—These are usually developed slowly and gradually. If at pasture the animal tends to isolate itself, and whether out or housed refuses food, or only takes small amounts and then stops feeding. There is also very little desire for water. There is evidence of abdominal discomfort as indicated by occasionally moaning, kicking forward with the hind legs, and occasionally lying down and rising again. The patient is usually dull and is rarely excited. Rumination ceases, and the milk yield is diminished. The abdomen appears rather distended, and respiration is blowing and somewhat difficult, owing to interference with the contraction of the diaphragm. The back is arched, and the hind legs spread or, where possible, standing at a lower level than the fore feet. This relieves the pressure on the diaphragm. In the early stages fæces are passed in small quantities and frequently, but as the condition gets more pronounced there is an absence of defæcation. On palpation of the abdomen on the left side the distended rumen can be felt, and its contents are of a firm doughy consistence. The hollow of the flank is obliterated, particularly on the left side, and, in fact, this area becomes a convexity instead of a depression. Percussion of the abdominal wall yields a dull flat sound, except in those cases which may be complicated by tympany, when resonance supervenes. In rare cases expulsion of the contents of the rumen by the mouth (vomiting) occurs. There is rarely any alteration of temperature. The pulse is sluggish and large, and the mucous membranes are dull or dirty looking.

Treatment.—Owing to the usually slow development of the condition, most of the cases are far advanced before the veterinary surgeon is called in. The condition is not so distressing as tympanites, nor is treatment so urgently required, but it is more difficult to relieve.

If the condition is complicated with tympanites, relief should be given as soon as possible by puncturing the rumen with the trocar and cannula. The instruments should be inserted at the most prominent part of the flank with a downward and forward direction. When gases cease to pass through the cannula the trocar should be reinserted and the two instruments withdrawn together. The object of the reinsertion of the trocar is to clear the mouth of the cannula of any solid food which might otherwise fall into the peritoneal cavity during the withdrawal of the instrument. All food should be removed and withheld for a day or two, and the abdomen should be kneaded or massaged to stimulate muscular activity of the rumen. This may be done with the closed fists pressing firmly into the rumen, and should be maintained for ten or fifteen minutes, principally on the left side. In extreme cases massage of the right side may also assist in restoring muscular activity. It should be repeated several times daily. Medicinally the best results are obtained by the administration of oily purgatives, together with some stimulant, and antispasmodic, such as oleum lini Oii. and oleum terebinthinæ ℥ii. Sulphate of magnesia ℔ss. to ℔i. may be given, and although useful is not so good, since its action is chiefly on the bowels rather than on the rumen.

Following the purgative the stomach may be stimulated and its tone improved by the combination of ammonium carbonate ʒiv. and powdered nux vomica ʒii. This should be repeated two or three times daily, being administered in a cold vehicle, such as milk or thin oatmeal or linseed gruel. The use of common salt is also frequently advocated, and may be administered in solution, or applied in small amounts to the back of the tongue, either alone or in the form of electuary. This stimulates thirst, and, as a result, the animal may voluntarily drink water, and so assist in reducing the consistence of the contents of the rumen. Wherever drenching is resorted to in the treatment of this condition, it must be practised with great caution, owing to the possibility of choking. For this reason some authorities prefer to use pilocarpine, eserine, or arecoline hypodermically, but the results do not appear so uniformly good, since they act more on the intestines than on the rumen. In mild cases relief can be procured in from one to three days, but in cases of severe impaction the condition may last for a week or more and still terminate in recovery. Improvement is generally indicated by a gradual returning desire for food, occasionally chewing of the cud, belching, and evacuation of the bowels.

In very obstinate cases it may be necessary to resort to the operation of rumenotomy for the removal of about two-thirds of the contents of the rumen (see p. 1025). When relief has been obtained, as is shown by the free passage of fæces, it is well to control the diet for a week or two, giving only succulent or demulcent foods in restricted quantities. The stimulant and tonic powder already recommended should be continued, with the addition of powdered gentian root ʒii.-ʒiv. twice a day for a week or ten days.

Tympanites of the Rumen; **Hoven, Blown, or Meteorism.**—This is a very common condition in ruminants, and consists in distension of the rumen with gases as the result of fermentation. The gases formed are usually carbonic acid gas (CO_2) and marsh gas (methane, CH_4). There is sometimes also a little sulphuretted hydrogen (H_2S), but the gas is only rarely offensive in odour. The condition may be caused either by the presence of excessive quantities of foods which rapidly ferment, such as roots (mangolds and swedes) and cabbages, particularly if they have been damaged by frost; sudden access to succulent green foods, such as young clover and aftermath, especially if the animal has been largely fed on dry foods previously. Various cereals rich in starch may also cause the trouble, such as wheat. It may also be due to defective muscular condition of the rumen, or defective secretions, both of which are conducive to stagnation of food in the compartment, thus favouring fermentation. When it is due to dietetic causes, it is a common thing to find several animals affected simultaneously. Cases due to intrinsic causes are generally single in occurrence. Tympanites may also be set up by interference with the natural eructations of gases (belching), and may thus occur as the result of choking. A similar result is produced when a ruminant becomes cast on its side from any cause, hence its occurrence in cases of milk fever in cows. Tympanites is most commonly met with in the late spring when the pastures are very wet, and in the autumn when animals are taken up from the pastures and fed on roots. Fresh clover is apt to cause a combination of tympanites and impaction on account of its succulent nature, and the tendency of ruminants to feed greedily on it when opportunity arises. Tympanites is most frequently associated with moist foods, while impaction is associated with dry foods.

Symptoms.—This condition may develop rather rapidly; the abdomen becomes quickly distended, and signs of abdominal pain develop, such as paddling movement of the hind feet, and frequent looking at the sides, kicking forward at the abdomen, and spasmodic swishing of the tail. Respiration is at first blowing, and as the condition advances becomes more and more difficult owing to the great pressure of the distended rumen on the diaphragm. The head is extended, and the nostrils dilated, and the mouth may be open, and the tongue protruded. On percussion of the abdomen, particularly of the left side, it is found to be very resonant, or drumlike, differing very markedly from the nature of the distension in impaction. The animal refuses all food (it may have been feeding right up to the moment when the condition developed) and rumination ceases. There may be some belching, but it is comparatively little in degree, and not sufficient to afford much relief. Some patients become very excited, and may be thrown into a state of perspiration. Fæces and urine are passed frequently and in very small quantities; this is owing to pressure of the distended rumen on the rectum and bladder. The pulse is accelerated, and larger than normal, owing to the pressure on the vessels of the abdominal area. The temperature is usually normal, unless the animal has knocked itself about, or is unduly excited. The mucous membranes become dull, and in advanced stages may become quite cyanotic. If the condition is due to choking, it is generally associated with excessive salivation and dribbling of quantities of saliva from the mouth. If relief is not afforded the patient is likely to die from asphyxia, owing to the great pressure on the diaphragm causing paralysis of that structure. The pulse gets smaller and weaker, and shortly before death is almost imperceptible, while the mucous membranes become still more deeply cyanosed. If the patient is standing he staggers and falls, and dies in convulsions.

The condition, although very serious in cattle, is still more dangerous in sheep. If seen early, however, it is probably not so serious a condition as impaction of the rumen, unless recurrent. It, however, calls for more urgent treatment, otherwise it is likely to prove fatal in a comparatively short time from asphyxia.

Treatment.—The first thing is to afford relief by removal of the gases from the rumen. In mild cases this can be accomplished by massaging the rumen with firm pressure, or by passing a rope round the body, and by tightening it, exerting pressure and assisting the expulsion of the gas. This may still further be assisted by drawing the tongue forward in the mouth or by inserting a gag into the mouth. A simple gag for this purpose consists of a thick straw band passed through the mouth and tied at the back of the head, behind the horns. In the more urgent cases, however, other methods must be resorted to. The simplest of these consists in drawing off the gas by means of a trocar and cannula, with a fair-sized bore, say not less than ¼ inch in diameter, or if flattened, not less than ½ inch by ⅕ inch. The instrument should be inserted on the left side at the most prominent part of the flank, which is usually about the middle of the triangle formed by the last rib, the transverse processes of the lumbar vertebræ, and the external angle of the haunch. It should be inserted downwards and forwards, so that at one effort it punctures the abdominal wall and the rumen. On withdrawing the trocar the gases escape through the cannula. Medicines are sometimes administered through a cannula directly into the rumen, and in this way can be given in a much more concentrated form than if administered by the mouth. It may be thought advisable to leave the cannula in position for a time in order to facilitate the escape of any further gases which may be produced, and, if so, it must be secured either to the skin by means of a suture, or by means of a tape or bandage passed round the animal's body. In the majority of cases, however, this is not necessary. In removing the cannula the trocar should be reinserted in order to remove any food material that may have passed into it, and so prevent it falling into the abdominal cavity and setting up peritonitis. The instruments should be kept sterilized, so that they are always ready for immediate use. Another method of affording relief consists in passing the stomach tube or probang, through which the gases will escape, and through which, if desired, medicines may subsequently be administered. For general purposes the former is the better method, as it is more rapid and attended by fewer risks.

Medicines administered are directed to the condensation of gases in the rumen, or to the reduction of fermentation and the prevention of the generation of further gases. For the former purpose powdered charcoal has been recommended, but since it has to be mixed with some liquid or semi-liquid vehicle for administration it is very doubtful whether it is of any service, since its powers of absorption are in this way obliterated. The administration of antizymotics is therefore preferable, and probably the best of many is oleum terebinthinæ ℥ii., which may be given in oleum lini Oii. or in soap solution or milk. Other useful agents are carbolic acid (ʒi.-ʒii. in water Oss.-Oi.), lysol, perchloride of mercury, chinosol, resorcin, and hyposulphite of soda (℥ii.-℥v.). To this may be added stimulants, such as carbonate of ammonia and nux vomica.

After-treatment consists in the administration of a purgative, if one has not already been given. A straw rope gag may also be inserted into the mouth to stimulate eructations. Attention should be paid to the diet, restricting those foods which have a tendency to ferment, and a course of stimulants and tonics should be administered, such as were recommended in the after-treatment of impaction (see p. 271). In recurrent cases, if the animal is in good bodily condition, it is well to consider the advisability to slaughter. The carcase would be fit for food provided that no noxious medicines have been given, such as the coal tar preparations and turpentine, which may have tainted the flesh.

Inflammation of the Rumen; Rumenitis; Alvitis.—Inflammation of the rumen is a very rare condition. The mucous membrane is apparently very resistant to irritants, but inflammation may, however, occur as the result of powerful irritant poisons, chemical or vegetable, and it is sometimes met with in the course of certain specific diseases, such as rinderpest and malignant catarrh. It is occasionally brought about by foreign bodies such as pieces of metal.

Symptoms.—The symptoms of this condition resemble those of indigestion, and consist of intermittent appetite, irregular rumination, and, in the case of milch cattle, diminished lactation. There is also a tendency to recurrent tympany, and sometimes vomiting. On manipulating or massaging the left side there may be a diffuse tenderness exhibited. The patient usually stands with his back arched, and the bowels are somewhat irregular. The condition is, however, rarely diagnosed, owing to the indefinite nature of the symptoms.

Treatment.—This depends upon the cause, if it can be discovered. If a known irritant is responsible its antidote should be administered. Tympany should be relieved by means of the trocar and cannula. If it is due to a foreign body, rumenotomy should be performed. In other cases an oleaginous purgative should be given, together with sedatives, such as carbonate of magnesia or bismuth or creta preparata. A counter-irritant may be applied to the left side, such as stimulating turpentine liniments, or a weak mustard plaster. The diet should be restricted, and of a succulent or demulcent nature. When the acute stages have passed a course of vegetable tonics should be administered.

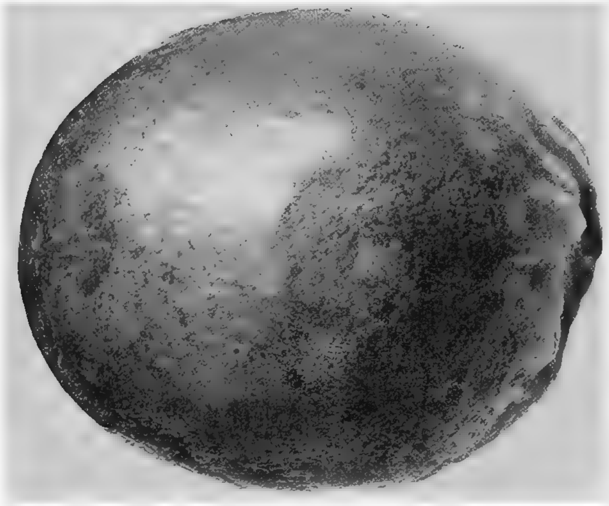


FIG. 68.—Hair-ball from an ox (natural size).

Hair-Balls in Cattle and Wool-Balls in Sheep.—Hair-balls and wool-balls are accumulations of hair or wool which have been swallowed generally by young animals. They may be brought about by the animals licking or sucking one another until a considerable quantity has been taken in. By constant movement in the rumen the hair or wool may become converted into a ball or sphere, while in other cases it assumes the shape of a short cylinder, whose diameter may actually exceed its length. Not infrequently the concretion of hair is coated with a deposit of salts, and has a highly polished surface. In other cases, the hair or wool is not coated, and the accumulation presents a surface rather like a closely woven felt. These balls may be single or multiple. In lambs it is not infrequently the case that the wool simply becomes matted together like an irregular mass of wet tow.

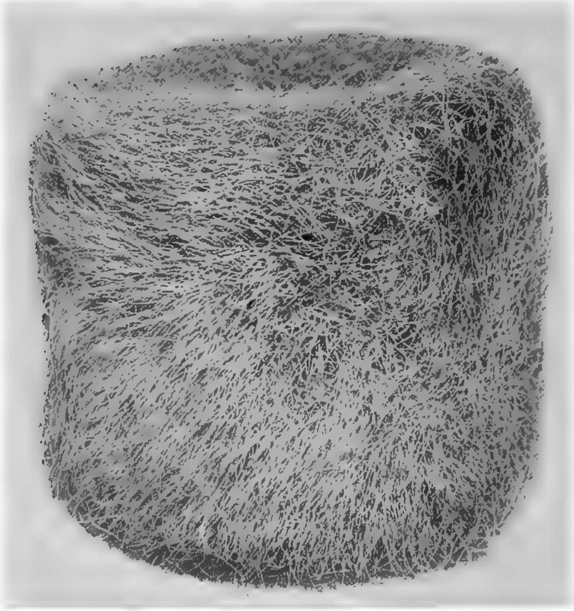


FIG. 69.—Cylindrical hair-ball from the rumen of an ox (natural size).

Symptoms.—These are very similar to those of recurrent indigestion, and are much more frequent in young animals than in adults. They may cause intermittent flatulence or impaction, according to their position. They may be found in the rumen, or in the reticulum or lodged in the œsophageal groove. Their frequent change of position is responsible for the intermittent character of the symptoms, but positive diagnosis is difficult or impossible.

Treatment.—If the animal is in good bodily condition slaughter is best resorted to. If, however, it is decided to adopt treatment, rumenotomy should be performed, otherwise the condition is likely to prove fatal sooner or later. It is not, however, invariably fatal, for occasionally hair balls are found in the rumen of adults on slaughter, having in all probability been responsible for recurrent attacks of tympanites during life.

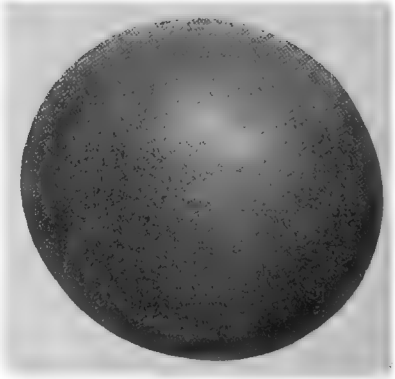


FIG. 70.—Concretion (1 of 8) from reticulum of a sheep (natural size).

Diseases of the Reticulum or Honeycomb

This, the second compartment of the stomach, is very rarely affected with diseased conditions, except such as may be due to the presence of foreign bodies. It is, however, occasionally inflamed during the course of certain specific affections, such as rinderpest.

Foreign Bodies in the Reticulum.—The reticulum of cattle is not infrequently the resting-place of foreign bodies, such as nails, which have fallen into it from the œsophageal groove, after having been swallowed. Other bodies found in the situation include various pieces of metal, stones, wood, and occasionally bone. If they are small they cause very little trouble beyond occasionally setting up indigestion, with recurrent tympany. In other cases, however, and particularly in the case of nails from 2 to 3 inches long, serious complications are likely to be set up, inasmuch as the piece of metal of this size may ulcerate the wall of the reticulum, and pass through in the forward direction towards the diaphragm, and ultimately find its way into the pericardium, setting up the condition known as traumatic pericarditis. The way in which this track is produced is a matter of some conjecture, and is undoubtedly very interesting. The size of the nail is of considerable importance, since neither larger nor smaller pieces of metal take this course. It is probable that by the movement of the wall of the reticulum the nail is as it were grasped, and the mucous membrane punctured, producing an ulcer which becomes infected. This deepens by virtue of the bacterial invasion and the continued irritation of the nail, and extension further contributed to by the movements of the diaphragm during respiration. By this extension a local patch of peritonitis is set up in contact with the diaphragm, and an adhesion is produced. The nail ulcerates its track through this adhesion, and is driven onwards through the diaphragm itself, which it pierces usually at the seat of attachment of the pericardium. It only requires a short step further for it to pass into the pericardial sac, and sometimes even into the heart wall. The condition therefore commences as one of ulceration of the reticulum, and ultimately develops into traumatic pericarditis.

Symptoms of Foreign Bodies in the Reticulum.—The symptoms as a rule are those of intermittent tympany and abdominal pain. The animal stands with arched back, and is constantly grunting. The appetite is capricious, and rumination often in abeyance. The bowels are irregular. The tympany is due to suspended eructations owing to the partial fixation of the abdominal wall, since movements of the abdominal muscles are likely to produce pressure on the foreign body and increased pain. Respirations are mainly thoracic. When the foreign body has passed through the diaphragm the symptoms are more or less completely changed. The abdominal symptoms become relieved, and thoracic symptoms supervene. The chest-wall becomes fixed, and the movements of the abdominal wall more pronounced. Tympany is less frequent, and the action of the bowels may be resumed. The pulse becomes very irritable, and the temperature elevated several degrees. As the fluid accumulates in the pericardium, as the result of pericarditis, dropsy may be set up, involving the front of the chest, the dewlap, and under the sternum. On auscultation of the cardiac area, friction sounds or the movement of liquid can be detected at each heart-beat. Such cases are invariably fatal. In other cases the foreign body may take a slightly different course, and may penetrate the liver, resulting in an abscess of that organ. This is usually fatal from internal hæmorrhage, with or without symptoms having been previously observed. Still again in other cases where the foreign body is long, such as the rib of an umbrella, it may ulcerate its way through the side, and ultimately, about the last rib, forms an abscess through which it may be removed, and the animal ultimately recover.

Treatment.—There is no treatment of any service, and if the condition is definitely diagnosed slaughter should be resorted to.

Diseases of the Omasum

Manyplies; Manyfolds; Psalterium; "The Book."—Diseases of this, the third compartment of the stomach, are extremely rare. In the normal condition food material present in this compartment is compressed between the muscular leaves, and is consequently always more or less dry. This condition by the layman is often wrongly regarded as a condition of impaction. Impaction of the omasum is possible, however, although very rare. It may occur when the organ is in a condition of atony, simultaneously with atony of the rumen and reticulum. The omasum is also occasionally the seat of inflammation.

Impaction of the Omasum.—When this condition occurs it is also known by the name of *fardel bound* or *maw bound.* It has also been included under the term *dry murrain.* It may be brought about by feeding on dry foods, by scarcity of water, and also as the result of paralysis or atony of the muscular folds of the organ. It is probable, however, that a considerable number of the cases which are diagnosed as impaction of the omasum on post-mortem examination are in reality normal conditions of the omasum.

Symptoms.—Impaction of the omasum may give rise to a wide range of symptoms making diagnosis somewhat difficult and uncertain.

In the earlier stages there are signs of abdominal pain. Grunting and grinding of the teeth are heard as in impaction of the rumen. The animal feeds capriciously at first, but later refuses all food. Rumination ceases, and lactation diminishes in milch animals. The temperature is unaltered. Fæces are passed in small quantities, but later there is a cessation of defæcation. In exaggerated cases the distended compartment is said to be felt on the right side, immediately behind the last rib, being very firm to the touch. It is impossible to palpate this organ in a state of health. Nervous symptoms may supervene. At first there is excitement with widely dilated pupils, staring eyes, and the animal may knock himself about considerably. Later, this stage of excitement passes on to stupor, and the patient staggers and falls and becomes semi-comatose and dies.

Treatment.—It is not easy to obtain good results in the treatment of this condition, largely owing to the difficulty of getting medicines into the affected compartment. An attempt may be made to reduce the consistence of its contents by the repeated administration of oleaginous purgatives, such as ol. lini Oii. together with ol. terebinthinæ ℥ii. This may be repeated in half doses daily. Alternatively saline purgatives may be given, but they are not to be recommended, since their action is mainly manifest on the small intestines, and not on the stomach. Probably the better method consists in the administration of arecoline or eserine gr. ii. hypodermically, and their effect is best seen after an effort has been made for a day or two to reduce the consistence of the ingesta. If the omasum can be felt it may be vigorously massaged several times daily. Instead of massage, it is sometimes advocated to apply a counter irritant, such as a cantharides blister or a mustard plaster on the right side over the region of the omasum. It is not advisable as a general rule, even in cases of excitement, to give sedatives, since they usually reduce the activity of the stomach walls. It is far better to administer a course of stimulants, such as carbonate of ammonia ʒiii. or ʒiv. and powdered nux vomica ʒi. or ʒii. twice daily. In any case, these stimulants should be adopted as soon as there is any indication of relief, as by a resumed activity of the stomach and bowels, and increased brightness of the patient, with some desire for food. Solid foods, however, should be withheld for several days, and a plentiful supply of water and demulcents, such as gruels, provided.

Omasitis; Inflammation of the Omasum.—This condition may be brought about by an irritant in the food being compressed between the leaves of the omasum, and thus brought into close contact with its mucous membrane. This organ, however, appears to be very resistant to irritants, and it is not easily inflamed. Omasitis may occur as a result of poisoning, with such irritants as yew, rhododendron, bracken, and arsenic. It may also be met with in certain specific diseases, such as rinderpest and malignant catarrh. It is very difficult to diagnose during life.

Symptoms.—These are principally signs of abdominal pain with grunting, grinding of the teeth, arched back, paddling with the hind feet, lashing of the tail, kicking forward at the belly, and general uneasiness. The bowels are irregular, the temperature may or may not be elevated. Assistance in diagnosis may be obtained by a history of the case. The animal may have had access to various irritant poisons, and on visiting the pastures there may be evidence of yew trees or rhododendron bushes having been nibbled, or yew clippings having been eaten. Positive diagnosis, however, may only be made on post-mortem examination. The mucous membrane of the leaves peels off readily, exposing a reddened surface, and evidence of diffuse vascular disturbance. If the mucous membrane peels off without such indication, it should be regarded as a simple post-mortem condition, resulting from commencing decomposition. Assistance may also be afforded by the discovery, both in the omasum and the rumen, of irritant substances, such as those enumerated above.

Treatment.—When diagnosed it is well to administer an oleaginous purgative (ol. lini Oii.) at once, in order to facilitate the expulsion of the irritant. If there is much pain it is well to administer an anodyne, such as opium (tinct. opii ℥ii.-℥iv., or pulv. opii ʒi.) or chlorodyne ℥ss.-℥i. or extract of belladonna ʒi. The diet should be of a succulent nature, consisting of cut grass, pulped roots, cabbage, and boiled linseed, or soaked linseed cake. When the acute stages are passed it is well to administer a course of vegetable tonics, such as gentian, nux vomica, and calumba, combined with caraway and aniseed.

Diseases of the Abomasum

The abomasum is the fourth compartment of the stomach in ruminants, and is the true digestive stomach. It may be the subject of inflammation, or it may be the seat of indigestion or dyspepsia. It is never impacted with solid foods.

Abomasitis; Inflammation of the Abomasum; Gastritis.—Inflammation of the abomasum may be brought about by irritants, such as arsenic, or irritant vegetable substances, which as a rule are contained in the food materials (simple gastritis). It may be superficial in character, or it may involve the deeper structures. The latter form may also be met with in specific affections such as rinderpest, anthrax. Another form of gastritis in ruminants is set up by various nematode worms (strongyles). This form is chronic in nature, and the symptoms of this parasitic gastritis differ very widely from that form due to irritants conveyed by food.

Symptoms of Simple Gastritis in Ruminants.—The symptoms may come on rather suddenly. If at pasture, the animals separate themselves from the rest of the herd, and stand with the back arched, are very uneasy, and show indications of abdominal pain. The appetite and rumination are capricious, but a continued desire for water may be evinced. The temperature varies, and may be anything from 102° to 105° or 106° F. The pulse is frequent and bounding in the early stages, but later becomes distinctly smaller, and in very acute cases almost imperceptible. The mucous membranes are somewhat injected. There is some degree of constipation, the fæces being passed in smaller quantities, and usually formed and mucus-coated. This may be followed by diarrhœa, particularly if the inflamed condition extends to the intestines.

Treatment.—It is well to commence first with a small dose of an oleaginous purgative, which should be followed by gastric sedatives, such as carbonate of magnesia, bicarbonate of soda, and bismuth carbonate in doses of ʒii.-ʒiv. of each. The administration of opium or chlorodyne is particularly advisable in those cases where diarrhœa has supervened. If the condition is suspected of being due to the contamination of food with arsenic as with sheep dip, then the antidote for arsenic should be given, namely, moist peroxide of iron (see "Arsenical Poisoning," p. 510). The food, as in other cases of inflammation of the alimentary tract, should be restricted, and demulcent in character (linseed gruel, oatmeal gruel, milk, boiled turnips, hay tea, cut grass, etc.). When the acute stages have passed, a course of vegetable tonics should be given, as recommended in the after treatment for omasitis.

Parasitic Gastritis in Ruminants.—(See p. 314.)

Pica; Depraved Appetite.—This is a peculiar condition of depraved appetite, which is met with in cattle, and occasionally in other animals such as pigs, horses, and goats. Cows in calf, and young cattle, are especially liable to develop pica. Affected animals have a tendency to lick and to gnaw almost anything with which they come into contact. They seem particularly disposed to eat earth and sand, soiled litter, and even excreta. The cause of the condition is not well understood. It is probably due to indigestion and dietetic errors which are difficult or impossible to specify. It has been suggested that an insufficiency of soda salts, or phosphates in the food, may cause it. It has also been asserted to be due to a nervous derangement, probably interfering with nutrition.

Symptoms.—When first noticed the animal may be in a good state of nutrition, but soon there is a loss of condition. The animal becomes somewhat restless, uneasy, and depressed. The appetite for normal foods is very capricious. The patient will almost at any time eat the abnormal substances already mentioned, and will also lick the clothes of the attendants, and gnaw at fixed objects to which they may be attached, such as the manger, partitions, etc. Later the animal becomes quite thin and wasted, and shows various symptoms of indigestion, such as intermittent tympany, irregular bowels and partially suspended rumination. The fæces become more or less dry and firm, although occasionally diarrhœic and offensive in odour. The mucous membranes become pallid, and the skin harsh and progressing towards hide-bound. If untreated, the animal may die from malnutrition and exhaustion, after a varying period which may extend over months. Depraved appetite often precedes osteomalacia in cattle, in which the bones become brittle and easily fractured.

Treatment.—First change the pasture, or if housed remove to another shed. Complete change of environment is necessary. Then administer a purgative, and follow with alkalies and bitter tonics. Murray recommends the following mixture:

Carbonate of iron . . .	℥iv.
Finely ground bone meal . .	℔. i.
Powdered gentian . . .	℥iv.
Common salt	℥viii.
Powdered fenugreek . .	℥iv.

These are to be mixed, and a heaped tablespoonful given three times a day. In addition to this he recommends three tablespoonfuls of powdered charcoal, to be mixed with the food three times daily. Special attention should be paid to the food, which must be generous and rich. It is also well to provide the animal with a lump of rock salt in his feeding-trough. Successful results are also recorded from the hypodermic injection of apomorphine gr. i.-iii. daily, for several consecutive days.

DISEASES OF THE STOMACH IN THE HORSE[1]

Inflammation of the Stomach; Gastritis.—Inflammation of the stomach of the horse is not a very common condition, largely owing to the selective habits of the horse over his food. The horse is, as a rule, by no means a gross feeder, and he will constantly refuse various deleterious food substances when hungry, even though there may be nothing else available. When gastritis does occur, it is generally due to irritant substances taken with the foods, either of a mineral or vegetable nature. Occasionally it is set up by parasites, and in other cases it is bacterial in origin. In the latter cases gastritis may occur as a complication of systemic catarrhal affections, such as influenza and purpura hæmorrhagica. Parasitic gastritis is exceedingly rare, and only occurs when the parasites are present in considerable numbers, such as bots (*Gastrophilus equi*), and spiroptera. Food substances responsible may be damaged and mouldy foods, and vegetable irritants such as yew, rhododendron and hawthorn berries. Foods contaminated with mineral poisons may cause gastritis, probably the commonest being arsenic, with which the food has become contaminated from sheep dips. Irritant medicinal agents insufficiently diluted may bring about similar effects.

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[1] See special chapter on the feeding of horses, p. 520.

Symptoms.—These are not very distinctive, except in acute cases. In *mild cases*, the appetite is capricious and discomfort and uneasiness are exhibited after feeding, only to pass away in an hour or so. There is loss of condition, and only slight if any elevation of temperature. The mucous membranes are dirty-looking, and the mouth may be somewhat furred, producing a sour odour of the breath. The bowels are irregular, with a tendency to constipation, the fæces being drier than usual, and passed in smaller quantities.

In *acute cases* of gastritis pain after feeding is much more pronounced, almost amounting to colic. The pain is more or less continuous for one or two hours, and then subsides, but recurs after the next meal. If the pain is very severe there is patchy sweating, particularly affecting the anterior half of the body. The temperature is usually elevated 1° or 2° F. in those cases due to irritants, but if bacterial, there may be a higher temperature up to 105° or 106° F. If the inflammation should extend to the intestines jaundice may arise, owing to the catarrh blocking the bile duct, also when the intestines are involved the tendency to constipation is likely to give way to diarrhœa. The paroxysms of gastritis are sometimes responsible for irregular peristalsis, which may result in twist of the bowel or volvulus. In these cases the colicky symptoms become continuous, and very severe, and terminate fatally instead of passing off in an hour or so.

Vomiting is not a frequent symptom of gastritis in the horse as it is in many species of animals; in fact, vomition in the horse in any circumstances is exceedingly rare, although in exceptional cases it may occur. This difficulty of vomition is probably due to the peculiar arrangement of the muscle fibres in the stomach wall and the termination of the œsophagus at the point where the latter leads into the stomach. In the majority of cases where vomition is suspected, the material is returned from a dilated œsophagus, and not from the stomach. In other cases, vomition in the horse is permitted as the result of dilatation of the stomach, particularly involving the cardiac portion, and the œsophageal opening.

Treatment.—It is best to withhold solid food for at least 24 hours, in order to give the gastric mucous membrane a rest, and the stomach tube may be used with advantage (see p. 305). The patient may be sustained with liquid nourishment such as linseed mashes, or linseed tea, oatmeal gruel, milk and eggs, and hay tea. In a day or two a little solid may be given, preferably boiled oats, and a small amount of well-steamed hay of good quality. Medicinally, since the condition is generally due to irritants, it is well to administer first a dose of the oleaginous purgative, such as oleum lini Oi.-Oiss. This also tends to correct constipation, which is so often associated with uncomplicated gastritis. It is not a good plan to give a violent purgative, since there is a probability of those producing superpurgation. Gastric sedatives should next be administered, such as chlorodyne ʒii.-ʒiv., or bicarbonate of soda, oxide or carbonate of magnesia in doses of ʒii.-ʒiv. These are best given in the form of electuary. If the pain is very acute, relief may be given by the hypodermic injection of morphia gr. iii.-gr. iv., or extract of belladonna ʒi., which may be administered with the electuary already mentioned. Objection is sometimes raised to the administration of opium and belladonna, owing to the interference with the peristaltic action of the bowels, but the advantages probably outweigh the disadvantages in cases associated with acute pain. As an alternative, however, chloral hydrate ℥i. may be given, but as this is somewhat irritant, especially to an already inflamed mucous membrane, it is best administered per rectum. In cases of fever the temperature may be reduced by salicylates, which may be given in the drinking water. If the condition is due to chemical irritants, such as arsenic, etc., the antidote should be administered. It is obvious also that further access to the irritant materials, whatever they may be, must be avoided. Horses should therefore be brought up from pasture, a complete change of diet should be ordered, and any foods which may possibly have been contaminated should be destroyed.

When the acute attack has passed away a course of tonics must be administered, such as a combination of nux vomica ʒi., with gentian and bicarbonate of soda, of each ʒii.-ʒiv.

Gastric Indigestion; Dyspepsia; Chronic Gastric Catarrh; Oxaluria.—This condition of imperfect digestion in the gastric compartment may be brought about by various causes. It may be due to defective secretion of gastric juice or to defective muscular power of the stomach wall. It is in many cases due to defective food materials and improper feeding. In this category are included those foods containing excessive quantities of indigestible fibre such as a too-liberal use of clover hay, sainfoin and lucerne hay, and straw; and also badly balanced foods having a too high nitrogenous ratio, as is occasioned by the excessive use of beans, peas, and grain (see chapter on feeding, p. 520). In other cases indigestion is due to imperfect mastication resulting from irregular or carious teeth.

Symptoms.—The horse slowly and gradually loses condition and gives evidence of inability to perform prolonged work with his usual vigour. His coat becomes dull and staring. Uneasiness, which may amount to subacute colic, occurs after feeding, but usually with an interval of an hour or more. This uneasiness is not so acute nor does it occur so rapidly after meals as in gastritis. The epithelium of the buccal mucous membrane tends to accumulate especially on the gums, giving the breath a sour odour. The visible mucous membranes are dull and anæmic, sometimes with a yellowish tinge. The temperature is not interfered with. The pulse lacks tone and is soft and sluggish. Horses frequently evert their lips and yawn, and also lick the walls. There is a tendency to constipation or irregularity of the bowels, and the fæces are often mucus-coated and are seen to contain elements of the food unaltered. There is also a frequent passage of flatus. The urine is yellowish on being passed, and on standing throws down a considerable precipitate. It is very rich in oxalates, particularly calcium oxalate, and this fact is responsible for the condition being called *oxaluria* by some veterinarians. The appetite in this condition is very variable, but in the majority of cases is well maintained, and in spite of this the loss of condition is progressive. In some cases brain symptoms are exhibited, the horse becoming very dull and sleepy, and may actually fall. This condition is sometimes called *stomach staggers* or *mægrims*, and is due to a nervous reflex action through the pneumogastric nerve producing either a temporary anæmia or venous hyperæmia of the brain. In rare cases of gastric indigestion fermentation may take place in the stomach, resulting in belching, or if the accumulation of gas is excessive and not relieved it may terminate in acute colic and rupture of the stomach.

Treatment.—It is well to commence with a mild purgative to remove any indigestible material present. The teeth should be examined, and any irregularities corrected either by rasping or shearing or extraction. There must be a complete change of diet, which must be of a nutritious and easily digested character, and not too bulky. The acute symptoms present should be relieved by chloral hydrate in preference to opiates. Medicinally one may have to experiment slightly in order to ascertain what agent will afford the best relief in the individual case. Pepsin may be given twice a day in ʒi. or ʒii. doses or hydrochloric acid dil. ʒi.-ʒii. Either of these may produce the beneficial results required, or they may be given in combination. In the majority of cases, however, better results are probably obtained by the use of alkalies which cause an increase in the secretions of gastric juice. These may with advantage be combined with vegetable tonics. A useful combination consists of ammonium carbonate, potassium bicarbonate, and powdered gentian, of each ʒii. with nux vomica ʒi. This powder may be given twice daily in a mash or in damped corn. Carminatives or stomach stimulants may be useful in some cases, such as caraway and aniseed or small doses of capsicum. When there is gastric tympany relief may be given by the administration of oleum terebinthinæ or chloral hydrate, which is both antizymotic and anodyne. If this fails it may be necessary to pass the stomach tube, which, however, must be done with great care in this condition, since if the horse should throw himself down it will probably bring about a rupture of the stomach.

The horse should not be expected to work in this condition, but properly regulated light exercise is helpful. Exertion and fatigue retard recovery.

Impaction of the Stomach in the Horse; Engorged Stomach; Stomach Staggers; Dilatation of the Stomach.—This condition may be brought about either by intrinsic or extrinsic causes. The intrinsic causes which may produce it are defective secretions, defective muscular power (atony) of the stomach walls, and stricture of the pylorus. Extrinsic causes consist of such foods as old dry innutritious grasses and those foods which have a tendency to swell in the stomach if improperly masticated, such as wheat and barley. These latter are likely to cause the trouble in animals that have not been accustomed to feeding on them, but the danger is averted if the corn is previously parched or boiled. Impaction is also predisposed to by an insufficient water-supply, and by animals bolting their food, as may result from irregular feeding, and in the case of greedy feeders or owing to the presence of diseased teeth. It is exceedingly bad policy to allow horses to fast for longer than usual periods. They have a comparatively small stomach, and should be fed in small quantities frequently. If they are kept without food for too long there is a natural tendency on the part of many horses to bolt their food greedily, with disastrous results. When food becomes accumulated in the stomach there is a tendency for it to swell somewhat, and if the stomach walls become distended excessively they become to all intents and purposes paralysed and are unable either to churn the food up for purposes of digestion or to pass it on into the intestines. It is extraordinary, however, how some horses will continue feeding after their stomach is already quite full. The writer has known a case where an impacted dilated stomach and its contents exceeded 100 lbs. in weight.

Symptoms.—These are ushered in by uneasiness which may amount to colic of varying degree. In other cases the condition is slowly progressive, the animal losing condition and becoming unthrifty from a preliminary stage of indigestion. When the stomach itself becomes distended there is interference with respiration, which becomes somewhat laboured owing to pressure on the diaphragm, and in some cases the patient has been described as sitting on his haunches to relieve the pressure on the diaphragm. This, however, is by no means a common or a diagnostic symptom, since it may also occur in other conditions, such as gastric tympany and in rupture of the diaphragm. If the stomach is greatly distended there may be a tendency to vomit. The horse is greatly distressed, and a greyish material may return from the nostrils. This is particularly the case if attempts are made to drench the horse. The pulse is usually full but soft, but there is no alteration of temperature. In some cases excitement occurs with abdominal pain indistinguishable from colic, while in other cases great depression, bordering on coma, is set up. The condition is exceedingly difficult to diagnose, and the true state is often not suspected until a post-mortem examination is made. In rare instances relief is obtained by vomiting, while in other cases death results from rupture of the stomach.

Treatment.—The treatment of this condition is by no means successful, and the majority of horses affected die as the result of it. It is useless to attempt to obtain relief by administering purgatives by the mouth, although one may be able to administer small doses of linseed oil and turpentine in the hope of it reaching the stomach and assisting to reduce the consistency of its contents. Liquor strychnia ʒii.-ʒiv. may be given hypodermically in the attempt to restore muscular tone and secretions, and also sulphuric ether ʒss. hypodermically as a diffusible stimulant. Hot blankets or a counter-irritant, such as a mustard plaster, may be applied to the abdominal wall and afford some relief. The stomach tube may be passed and an attempt made to wash out the stomach (see p. 279). If this is not successful, eserine or pilocarpine grs. i.-ii. may be administered, or the two may be given in combination in half doses. This must be regarded as a drastic measure, and if it does not afford relief may result in rupture of the stomach. Copious hot enemata should be administered with a view to stimulating peristaltic action and relieving pain. All food material should be withheld, and it is often necessary to change the bedding to some material such as moss litter or sawdust, which the horse is not likely to eat. A pail of water should be left constantly in a low position, in the hope that the patient will help himself to it from time to time. If the horse recovers from the impaction it will necessitate considerable care in the future. He must have regular feeding and a liberal allowance of water. His food should be soft, long foods being withheld. His teeth should be examined and attended to if necessary. A course of vegetable tonics should be prescribed. If the impaction has been due to intrinsic causes a recurrence is very likely to occur.

Gastric Tympany in the Horse.—This condition is due to fermentation of foods in the stomach which become distended with gases. It therefore almost entirely depends upon the nature of the foods. Young clover and vetches fed to horses in the early part of the year in excessive quantities, and especially if they have been kept fasting longer than usual, is a very likely cause. It may also be produced by feeding on barley and wheat and other soft foods. Other causes are frozen and diseased roots. Gastric tympany may or may not be associated with intestinal tympany.

Symptoms.—These vary considerably in horses, depending upon the extent of the distension. They occur, as a rule, an hour or so after feeding. There is considerable pain and distress, pawing, sweating, and blowing respirations. The horse will frequently cringe as though to throw himself down, but, as a rule, refrains and rises again. Sometimes he goes down and rolls. Occasionally there are eructations of gas from the mouth, which afford a little relief, but it is rarely sufficient to have much effect. The abdomen becomes somewhat swollen and tense, and the pressure of the distended stomach on the diaphragm causes dyspnœa or difficult respiration. As in the case of impaction of the stomach, depression of the hind quarters, almost amounting, if not quite, to sitting on the haunches, may occur, evidently in the attempt to reduce diaphragmatic pressure. If relief is not afforded fairly soon, rupture is likely to follow.

Treatment.—If it were possible the best plan would be to directly remove the gases from the stomach, as in the case of tympanites in cattle, but owing to the position of the comparatively small stomach and the fact that it does not come into direct contact with the abdominal wall, this course is impossible. If the horse is very easily handled an attempt to pass the stomach tube may be successful, but this must be done with great caution, owing to the danger of the patient throwing himself down and producing rupture of the stomach. The administration of antizymotics to reduce the fermentation should be resorted to, and probably the two best agents are oleum terebinthinæ ℥ii. and chloral hydrate ℥i.-℥ii., both of which have additional diffusible stimulant action, and assist in promoting the expulsion of the gases along the intestinal tract. Reeks recommends the combination of sulphate of iron ℥i. with liq. ammon. fort. ʒiv. in water Oii., to be followed up with terebene or turpentine. If relief is not effected by this means it should be followed by the hypodermic injection of eserine or pilocarpine. This must, however, be regarded as a drastic resort, and is not without danger, as it may easily cause rupture of the stomach with fatal results. After-treatment consists in careful dieting with a combination of alkalies and vegetable tonics, such as are recommended for gastric indigestion.

Rupture of the Stomach.—This condition is rarely met with in any other species than the horse. It may result from acute tympany or from acute spasms during excessive impaction of the stomach. In the latter cases the actual rupture in all probability takes place as the result of an attempt to empty the stomach by vomition. Rupture may also occur as the result of violence during a fall soon after a meal, or when the stomach is otherwise distended.

Symptoms.—If rupture occurs during tympany or impaction the symptoms of those conditions which have been observed suddenly cease and the animal temporarily appears relieved. This may give rise to a false impression on the part of the owner that the horse is much better. Shortly afterwards, however, profuse sweating occurs associated with shivering fits or rigors. The extremities become cold, respiration is blowing, the pulse assumes a running-down character, getting gradually smaller and weaker until almost imperceptible. Peritonitis rapidly supervenes with the distension of the abdomen, tenseness of the abdominal muscles, and pain on pressure of the abdominal walls.

It has been sometimes asserted that as the result of rupture of the stomach vomition occurs, but it is far more probable that the converse is the case, namely, that the rupture itself may be preceded by vomition, with the escape of fluid or semi-solid ingesta by the nostrils. When the rupture has actually occurred the escape of contents of the stomach during spasmodic contraction will undoubtedly take place through the channel of least resistance, namely, through the rent. It is almost inconceivable that with a rupture present food material can be expelled through the œsophagus, when one realizes how extremely difficult the procedure is, even in the absence of any rupture of the wall of the stomach. When the rupture is extensive death may occur in from half an hour to several hours. In other cases where a slight rupture only is present, peritonitis is set up and the patient may last one or two days.

It is sometimes a matter of importance to decide whether the rupture of the stomach may have occurred post mortem, and this can readily be done by reference to the edges of the ruptured viscus. If the rupture has occurred before death there will be marked evidence of vascular disturbance with hæmorrhage. On the other hand, when the rupture is post mortem there is no sign of blood, and the torn edges are usually pallid. Post-mortem rupture may, of course, occur as the result of continued fermentation and gas formation after death.

Foreign Bodies in the Stomach of the Horse.—Foreign bodies are rarely met with in the horse except in the few cases which may be described under the heading of *pica*, the condition of depraved appetite corresponding to a similar condition more frequently met with in cattle. The foreign substances may consist of earth or sand, or various pieces of metal. Occasionally concretions are met with, composed chiefly of dirt and oat-hair and oat-husks, stuck together by the aid of mucus. These concretions, however, as is the case with other foreign bodies, are more frequently met with in the large intestines. They are usually only discovered on post-mortem examination and give rise to no symptoms by which they can be diagnosed.

DISEASES OF THE STOMACH OF THE DOG

Gastric Indigestion.—This condition in the dog may be due to improper foods, or it may be due to defects in the quantity or quality of the gastric juice. The ease with which a dog can empty his stomach by vomiting and so relieve himself of the presence of indigestible materials in the stomach, accounts for the fact that this is not a very common cause of indigestion in the dog. A condition corresponding largely in effects with gastric indigestion not infrequently occurs in young excitable dogs that are allowed to frolic or become otherwise excited shortly after feeding. In many of these cases the condition is no doubt due to nervous causes and may be described as *nervous dyspepsia.*

Symptoms.—These are generally observed an hour or so after feeding. There may be some preliminary dulness or uneasiness or not, and in many cases vomition is the first symptom observed. The dog will frequently consume the vomit again almost immediately. There are frequent eructations of offensive gases from the stomach. Depraved appetite is a common symptom, dogs eating sometimes most objectionable and repulsive substances. These in turn aggravate the condition of indigestion so that a "vicious circle" may be thus set up. The mucous membranes are pallid and dirty-looking, while in the mouth there is frequently some coating or furring of the teeth and tongue, rendering the breath rather offensive. The temperature is not interfered with. The bowels are irregular, with a tendency towards constipation. There is a passage of profuse quantities of urine from which a yellowish white deposit is formed on standing. The condition is associated with considerable thirst, and after drinking quantities of cold water vomition generally occurs. Affected dogs have a tendency to stretch themselves out on the sternum with the abdomen in contact with cold stones, but this attitude is more frequently adopted in acute gastritis. Hiccough is often observed, and in highly nervous dogs there may be spasm of the diaphragm with great dyspnœa and very exaggerated thoracic respiration. At first this spasm is very distressing to the owners of the dog, but it passes away after a few seconds and apparently leaves no ill effects. If the indigestion is continued for long there is great loss of flesh, and anæmia and debility are set up.

Treatment.—It is advisable first to administer an oleaginous purgative, such as oleum ricini with or without the admixture of syrupus rhamni. Alternatively hydrargyri subchloridum (calomel) may be administered. Great attention to the feeding is necessary, and it is often a matter of experimentation to find what particular diet is most suitable to the individual case. In a general way a flesh diet is best, such as minced beef or boiled sheep's paunch, or liver partially cooked, or a little rabbit. Starchy foods are better avoided, so that, for the time being at any rate, biscuits and potatoes should be withheld. Water supply should be curtailed; in fact it is often best to completely withhold water, substituting for it barley water or milk, with the addition of a little lime water or Vichy water. In any case, boiled foods and liquids should be given in small quantities during the acute stages of the indigestion. Medicinally again, a certain amount of experimentation is necessary before one ascertains the best agents for the particular case. For general purposes the following may be used :

Bismuth carbonate gr. iii.-gr. x.
Sodium bicarb. gr. v.-gr. x.
Spts. ammon. aromat. m. x.-xxx.
Tinct. nucis vom. m. ii.-x.
Aqua chloroformi ad ʒii.

To be administered twice daily in a tablespoonful of cold milk.

Other useful agents are tinct. or infusion of gentian or of calumba and compound tinct. of cardamoms. In some instances the above do not afford the necessary relief, and then pepsin gr. v. and dilute hydrochloric acid m. v.-x. may be tried. The patient should be given regular exercise, which should not be excessive, and in any case not within an hour after feeding.

Impaction of the Stomach of the Dog.—This condition is not very likely to occur in the dog, owing to the ease with which he can vomit and so relieve impaction voluntarily, and also owing to the fact that the nature of his food is rarely such as will swell after ingestion. It occasionally occurs as the result of the ingestion of bones, particularly of poultry and game, by greedy dogs. In rare cases impaction may be due to depraved appetite resulting in the ingestion of quantities of grass and straw, or other foreign bodies. The presence of numerous worms may be a contributing cause. Sometimes it is the result of an obstruction of the pylorus by a foreign body, such as a ball which may have been swallowed inadvertently. Fermentation is not likely to occur in the stomach of the dog, and consequently rupture of the stomach is rare, unless it is directly brought about by a sharp foreign body, such as a bone or skewer.

Symptoms.—Symptoms usually occur rather rapidly, and generally commence with urgent attempts at vomiting. If this is successful the condition is immediately relieved and there may be no further symptoms of anything wrong. In other cases, however, attempts at vomition are in vain, and the distress may become rather acute. On palpation of the abdomen the impacted mass may be found in the distended stomach. As a rule, there is no alteration of temperature. The bowels are irregular, frequently diarrhœic. In some instances great nervous excitement is produced, and the patients run about almost as if they were mad, and owing to this, mistaken diagnosis of rabies has sometimes been made. Unless treatment can be afforded fairly soon, gastritis is likely to supervene with fatal results.

Treatment must be adapted to the peculiarities of the individual case. If impaction is due to any materials other than sharp bodies, a general emetic may be first administered, such as apomorphine hypodermically (gr. $\frac{1}{20}$ to gr. $\frac{1}{8}$), and if this is ineffective a small dose of oil may be administered, combined with a little ether to assist its retention. If the impaction is due to sharp bodies, such as poultry or game bones, or to a ball or other foreign body, surgical relief (laparo-gastrotomy) must be resorted to. Subsequently attention must be paid to regularity of feeding and the nature of the food. Bones should be avoided and any flesh foods should be minced. It is often a good plan to withhold solid foods for a day or two, allowing only milk or gravies or oatmeal porridge. Attempts should be made to restore action of the stomach wall by means of gastric tonics and sedatives, such as are recommended in the treatment of gastric indigestion in the dog.

Gastritis in the Dog.—In spite of the fact that a dog can so readily relieve his stomach of the presence of any irritant materials which he may have taken, yet he is more frequently the subject of acute inflammation of the stomach than any other of the domesticated animals. This may be due partly to his greedy feeding propensities and the fact that he will often ingest food substances containing chemical and other irritant poisons which may have been incorporated with it and laid down for the destruction of vermin. Another reason for the frequency is the fact that gastritis in the dog is often specific in character, being either primary or met with in the course of such affections as distemper and canine typhus. A superficial catarrhal gastritis may also be caused in puppies by parasites present in the lumen of the stomach, such as the *Ascaris marginata*, whose normal habitat is the intestine. Two forms of gastritis may be recognized in dogs, viz. subacute gastritis and acute gastritis.

Subacute Gastritis in the Dog or Gastric Catarrh.—This is a rather common condition in certain classes of dogs, and is perhaps most frequently met with in bull-dogs. The symptoms set up very closely resemble those of indigestion, to which the reader is referred. Vomition, is, however, more persistent than in indigestion, and diarrhœa is more frequently met with. The wasting and loss of flesh are more marked, and there are greater daily variations in the temperature. The chief danger from this condition is the frequency with which it may suddenly assume the characters of acute gastritis with rapidly fatal results.

The treatment of subacute gastritis in the dog is the same as for gastric indigestion.

Acute Gastritis in the Dog.—The serious nature of acute gastritis in the dog cannot be overestimated, owing to the very rapid course which it may assume, not infrequently proving fatal within twenty-four hours of the onset of symptoms.

Symptoms.—The first thing observed is usually vomiting immediately after food, together with marked thirst, especially for cold water, which is invariably vomited immediately after being taken Later vomits are likely to consist of slimy mucoid substance, sometimes white and frothy, at other times yellowish or green owing to the presence of bile. In acute cases, and particularly if the gastritis has been long in existence, the vomit is often blood-stained, and when due to canine typhus is chocolate-coloured and extremely offensive in odour. In uncomplicated gastritis constipation occurs, but if the inflammation extends to the intestines it will be followed by diarrhœa which is offensive in odour, and may or may not be blood-stained. The pulse in the early stages is somewhat irritable and small, but later becomes almost imperceptible. There is great tendency for the patient to lie with his epigastrium on cold stones. Pain is evinced on pressure of the epigastrium, and the patient frequently yelps if taken up with the hands passed under the body. On palpation of the epigastrium pressure on localized areas may reveal evidence of circumscribed pain. This usually indicates the situation of ulcer of the stomach. There is a very rapid loss of flesh and rapidly progressive weakness. The teeth and mucous membranes of the mouth become coated and offensive in odour, and the tongue badly discoloured, almost black. The temperature in the early stages is elevated several degrees, but in the later stages where collapse is imminent the temperature becomes subnormal. It is then rapidly followed by a cold body surface, cold extremities and lips, general collapse and death. When gastritis is due to chemical irritants it is often associated with stomatitis, as in the case of phosphorus poisoning. The vomit in the latter case can be distinguished from other forms of gastritis by being luminous in the dark (see " Phosphorus Poisoning," p. 512).

Treatment.—Gastric sedatives are called for in this condition in order to relieve the irritability of the stomach and to reduce the vomiting. The best results are usually obtained by the administration of bismuth carb. or bismuth subnitrate gr. v.-gr. x., three or four times a day, placed on the back of the tongue in the form of a powder and washed down with a teaspoonful of milk or albumen water, or administered in some mucilage such as tragacanth. Salicylate of bismuth, carb. of magnesia, bicarbonate of soda and chloretone are also very useful given in similar doses. Sometimes the combination of chlorodyne with either of the above affords better relief. Small doses of cocaine or of hydrocyanic acid relieve the irritability of the gastric mucous membrane, and are sometimes serviceable when other means fail to check the vomiting. Gastric disinfectants are frequently very useful, such as quinine, chinosol, permanganate of soda or potash or calcium, boracic and salicylic acids, etc. If constipation is very marked, relief should be given by means of enemata either of soapy water or glycerine and water or warm oil. Purgatives are best avoided owing to the danger of setting up diarrhœa, which is very difficult to check, and thus aggravating the debility and exhaustion. Hot applications to the abdominal wall are very serviceable, both relieving the pain and maintaining the body temperature. Water should be assiduously withheld in spite of an evident craving for it on the part of the patient. This thirst may be relieved by small enemata of normal saline solution, or by a very small allowance of albumen water or barley water, and a little ice. The question of food is of considerable importance, since it is essential that the strength of the patient should be maintained. There are marked divergencies of opinion as to which is the best type of food in such cases. Probably the best results are obtained by beating up the white of an egg in a teacupful of milk with the addition of a dessertspoonful of brandy, and administering this in very small quantities not exceeding one or two teaspoonfuls at a time for small dogs or tablespoonfuls for large dogs. This mixture is retained in a majority of cases when most others are vomited. Other foods which may be used alternatively consist of milk and lime water, various meat essences of which Brand's Essence of Beef is probably the best, also given in small doses. At other times farinaceous substances such as arrowroot or cornflour or Benger's Food are found to be more useful. In any case the cardinal rule is for the administration of nutriment in very small quantities and frequently. When none of these substances can be retained nutrient suppositories must be resorted to and many practitioners prefer to use them throughout the acute stages of the affection. During convalescence it is necessary to put the patient on a course of tonics similar to those recommended for gastric indigestion.

DISEASES OF THE STOMACH IN SWINE

Gastritis.—Inflammation of the stomach of the pig may be brought about by irritants in the foods, such as adulterants which are frequently found in pig meals, also foreign bodies, such as stones, pieces of wood, etc.; various irritant poisons cause gastritis in the pig, such as salt or saltpetre, arsenic from sheep-dip, and copper from preparing pigs' food in a copper vessel in which it is allowed to stand and become sour. Gastritis may also be due to bacterial causes, and may thus be met with in certain specific affections, such as swine fever, swine erysipelas, and occasionally anthrax and tuberculosis. Occasionally it may be due to the presence of worms (*Ascaris suis*).

Symptoms.—If there are several pigs feeding together it is probable that several will be affected simultaneously. Symptoms come on suddenly with some severity. There is vomiting, particularly after food, and great thirst. The patient stands with arched back and tucked-up abdomen, indicating abdominal pain, and as the condition advances shows no disposition to feed. The temperature is usually somewhat elevated (103°-105° F.) according to the cause of the condition. In the early stages there is usually constipation, and this is generally followed by diarrhoea, which may or may not be blood-stained or black. The skin is usually discoloured, being red and frequently carrying a rash. In quite young pigs brain symptoms may be observed, especially when the condition is due to worms, the animals having convulsions and muscular twitchings.

Treatment.—If the pigs are in good condition it may be advisable to slaughter, and if otherwise fit use the carcase for food. If this course is not adopted an oily purgative should first be given, followed by stomach sedatives such as chlorodyne and the carbonates of sodium, potassium, magnesia, and bismuth. There should be a complete change of food, and during the acute stages linseed tea or barley water or milk should be given in the place of solid foods.

Indigestion in the Pig.—This condition is not a very common occurrence, owing, as in the case of the dog, to the ease with which indigestible materials may be removed from the stomach by vomiting. It is, however, met with in pigs that are badly nourished and have only dry and coarse foods available as when out at pasture with little or no additional artificial feeding. In such cases indigestion is set up by the simultaneous presence of the indigestible material together with lack of secretions and reduced gastric tone. The stomach of the pig may also be the seat of curious elongated concretions slightly constricted in the middle, and with a coating of bristles which stick out loosely at the end, almost like a large worn-out shaving brush. They are not diagnosed during life, but give rise to symptoms of indigestion.

Symptoms.—Vomiting after feeding is fairly constant, but as a rule only a portion of the contents of the stomach is evacuated. The pigs lose condition and are apt to become pallid and anæmic. There is considerable thirst, and the bowels are irregular—one day more or less constipated and the next day loose. The pig has a tendency to lie about in a lackadaisical manner, and to bury himself under straw bedding if available. He objects to being disturbed. Not infrequently there also arises a discoloration of the skin, particularly noticeable behind the ears and under the belly. Swine appear to be peculiarly predisposed to skin rashes in any condition of ill-health, hence this occurrence not only in local affections but also in most specific diseases. If the indigestion becomes very bad and lasts long, marked weakness arises particularly of the hind-limbs, resulting in a rolling gait. In the case of young pigs, fits or convulsions may occur.

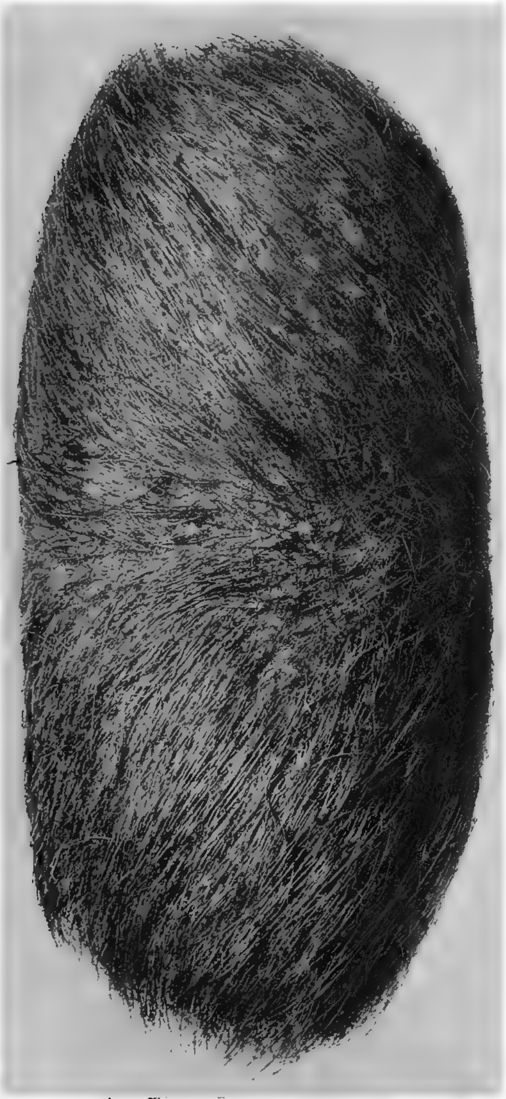

Fig. 71.—Concretion from the stomach of a pig ($\frac{3}{4}$ actual size).

Treatment.—It may be as well to consider first the general condition of the animal. If he is a fat pig, slaughter is probably the best thing to do. If, on the other hand, the animal is otherwise too valuable, as for breeding purposes, treatment may be adopted. If treatment is decided upon a laxative should be administered, and if the pig is feeding he will usually take readily a dose of oil in his food. If he is not feeding it may be necessary to administer either oil by drenching or a combination of calomel gr. xv.-xxx. and jalap ʒiv.-vi., which is best mixed with treacle, and inserted in the mouth by means of a flat piece of stick. Subsequently the condition can be improved by the daily administration of bicarbonate of soda ʒi.-ʒii., with powdered gentian ʒss.-ʒi., once or twice daily. If vomiting is persistent it may be checked by means of carbonate of bismuth gr. x. or gr. xv. or chloretone in a similar dose. Attention should be paid to the food and coarse materials avoided, and if the animal has been running out at pasture he should be brought up and housed, and fed with easily digested less bulky foods.

STRICTURE OF THE PYLORUS; OBSTRUCTION OF THE PYLORUS

These conditions may occur in any species of animal but are not at all common. The cause of stricture is not well understood, but it is probably a sequel in most cases to an injury which may have been inflicted by some foreign body taken in with the food. Obstruction may be due to mechanical causes such as a stone or a ball that may have been swallowed, particularly in dogs, or to tumours pressing on the pylorus from the outside.

Symptoms.—These are usually associated with chronic indigestion and distension or dilatation of the stomach. This dilatation is generally recurrent and becomes progressive, until after several attacks the stomach may be considerably enlarged and the abdomen pendulous. There are signs of abdominal pain, varying in intensity according to the severity of the distension. Respirations are interfered with owing to the pressure of the distended stomach on the diaphragm. In dogs there is frequently repeated vomiting and symptoms suggestive of chronic gastric catarrh with abdomen tucked up, while in rare cases of obstruction by a foreign body the dog may become quite frantic and give rise to a mistaken diagnosis of rabies. The condition is likely to prove fatal eventually, either from rupture of the stomach or from persistent vomiting and exhaustion.

Treatment.—Stricture of the pylorus cannot be removed, and treatment must be directed to minimizing the effects of the condition. In the case of the horse, food should be withheld for about twenty-four hours, but plenty of water offered and left constantly with him. A dose of oleum lini Oi. and oleum terebinthinæ ℥i. may be administered to reduce the tendency to tympany. When the condition is relieved any animals subject to it should be fed frequently with concentrated and not bulky foods, and in the case of the horse soft foods are found to be the best. When mechanical obstruction occurs relief can only be afforded by surgical means (laparo-gastrotomy).

DISEASES OF THE INTESTINES

Enteritis or Inflammation of the Intestines

Enteritis is a fairly common affection of all the domesticated animals and various forms of the disease are recognized. Superficial inflammation is frequently spoken of as *catarrhal enteritis* or *muco-enteritis,* while inflammations involving the deeper structures of the intestines are called *phlegmonous enteritis.* When affecting the small intestines it is known as *duodenitis*, *jejunitis* or *ileitis* according to the region involved. In the large bowels, inflammation of the cæcum is called *typhilitis*; inflammation of the colon, *colitis*; and inflammation of the rectum, *proctitis.* Except in the case of ruminants the large intestines appear to be more frequently inflamed than the small intestines.

Enteritis in the Horse.—Inflammation of the bowels of the horse is frequently met with, the intestinal mucous membrane being apparently more delicate and more susceptible of irritation and injury than that of any of the other domesticated animals.

Causes of Enteritis.—The general cause of inflammation of the bowels is an irritant of some kind. These irritants may be in the nature of improper food and errors of diet, as in the case of excessive feeding with new meadow hay, clover hay, or sainfoin or lucerne hay and oat-husks. Excessive feeding on beans may also set up the condition. Irregularity of feeding with solid foods is a predisposing cause. Mechanical causes may operate, as in the case of foreign bodies like pieces of wire or nails which may have been taken in with the food. Chemical irritants may also cause inflammation of the bowels under similar conditions as they produce gastritis. Enteritis in the horse is very frequently of bacterial origin, and may accompany such diseases as anthrax, influenza, purpura hæmorrhagica. The horse is also subject to infestation with a number of vicious parasites which inhabit the bowel and not infrequently set up inflammation of a serious nature.

Catarrhal Enteritis or Acute Intestinal Catarrh—*Symptoms.*—Symptoms of this condition are generally ushered in by some loss of appetite with rigors or shivering fits. At first there is some constipation and the fæces are mucus-coated, but this soon gives way to diarrhœa, soft fæces being passed frequently and in small quantities. Excessive rumbling of the bowels (*borborygmus*) may be heard. When the lower part of the large bowel is involved, and particularly in proctitis, straining or tenesmus is very frequent. The pulse is usually small and hard in the early stages, but later becomes larger and softer. The temperature rises several degrees and may be anything from 102° F. to 105° F. The body surface and extremities are rather cold, particularly if a large area of the bowels is affected. The horse may exhibit symptoms of dull but more or less continuous abdominal pain, but this is very varying in degree. The pain and restlessness are generally indicated by the patient walking round and round the box and looking round at his side. Respirations are not appreciably interfered with unless the attack is very acute, in which case there may be some fixation of the abdominal wall with blowing thoracic respiration. If the catarrh extends to the bile ducts, jaundice may be set up with yellow mucous membranes and a sudden acute rise of temperature. If the condition lasts long the abdomen and flank become very tucked up. Catarrhal enteritis may last a varying time from twelve or twenty-four hours to seven or eight days in acute cases, but in subacute cases it may last for several weeks. In fatal cases a patient gets gradually weaker and thinner, and has a rolling gait, and may be unable to stand. The temperature becomes subnormal and death occurs from exhaustion.

Treatment.—An effort must be made both to reduce the pain and the activity of the bowel in order to relieve this condition. Opium and its derivatives will effect both these aims. The hypodermic injection of morphia gr. iv.-gr. vi. is of service in acute cases. If opium is given it should be in fairly large doses such as pulv. opii ʒi. or tinct. opii ℥iii. or ℥iv. Chlorodyne in ℥ss. to ℥i. doses is similarly very useful. Some practitioners for various reasons object to the use of opiates and prefer to rely upon repeated doses of chloral hydrate. This agent undoubtedly relieves the pain but has not the same advantageous effects of reducing the secretions and peristalsis. If the pain and the diarrhœa are not very acute, very good results can be obtained by the use of salicylates or salol ʒii.-iii. doses, which may be administered in electuary and repeated several times daily. In cases where collapse threatens it may be necessary to administer stimulants such as carbonate of ammonia ʒii.-iv., in bolus, or sulphuric ether or caffeine hypodermically. When the acute stages are passed a combination of carbonate of ammonia, carbonate of iron and nux vomica may be administered. The patient should be well rugged up and bandages applied to the legs, and should be kept comfortable and warm in a well-ventilated box. His diet for several days should be demulcent in character, consisting of a little boiled linseed or boiled oats and a very small amount of steamed hay of good quality. Water should be tepid and curtailed in quantity.

Phlegmonous Enteritis in the Horse.—This is deep-seated inflammation of the bowels, involving the submucous and muscular coats and causing considerable thickening of the bowel wall. It frequently gives rise to a croupous exudate, which coagulates on the surface of the bowels, and is sometimes called *pseudo-membranous* or *diphtheritic enteritis*. This form may be brought about by corrosives or powerful irritants, such as arsenic and castor-oil seeds which contaminate the food, and it is met with in anthrax and in purpura hæmorrhagica. It may also be produced by worms, particularly the immature forms of sclerostomes which are met with in the bowel wall. This form of enteritis may be localized or very extensive.

Symptoms.—When the condition is due to corrosives or bacteria there is a tendency to abdominal pain of varying intensity, but generally more severe than in the case of muco-enteritis. The horse walks round and round the box, paws with his fore-feet, and appears very distressed. Respirations are mainly thoracic and are accelerated. The pulse is frequent and hard and small in the early stages, and later becomes fuller during the crisis, but in fatal cases it is ultimately very small and almost imperceptible. The temperature varies from 103° F. to 106° F., while the body surface and extremities are cold, particularly in the early congestive stages. In many cases the patient breaks out into patchy cold sweats. The fæces are passed frequently and in small quantities, and are usually soft in consistence. In the early stages they may be a little firm and covered with mucus. When the large intestines are affected diarrhœa supervenes, and in intense cases the fæces may be blood-stained. The passage of fæces affords no relief of the symptoms. This fact helps to differentiate the condition from impaction of the bowel. Phlegmonous enteritis usually assumes an acute course of varying length. It may last a week or a month, and in either case terminate in death or recovery. Prognosis, however, should be guarded, as it is quite impossible in a large number of cases to foretell the termination. On recovery a long period of convalescence must be allowed.

When enteritis is due to anthrax in horses the symptoms of pain are very intense, resembling acute colic, and the condition proves fatal within forty-eight hours. When enteritis occurs during the course of purpura hæmorrhagica it is very serious and usually has a fatal termination.

When phlegmonous enteritis is due to sclerostomes an entirely different train of symptoms is set up. It is much less acute, and abdominal pain is not usual. The chief symptoms in this form consist of progressive emaciation, debility, and anæmia, with persistent diarrhœa of offensive odour supervening. In the excrement the small mature parasites may often be observed.

Treatment.—If there is good reason for suspecting any particular corrosive poisoning the antidote should be immediately administered. One should then direct attention in the first place to the relief of the symptoms, to reduce pain and to diminish peristalsis in order to give the bowel wall as much rest as possible. For this purpose opium and its derivatives undoubtedly afford the best results. These may be combined with intestinal astringents such as catechu, tannin, sulphate of iron, acetate of lead, and tannoform. Salol is also useful as an intestinal antiseptic. Stimulants and febrifuges may also be required, and recourse may be had to ammonia and quinine, together with iodide of potassium. The animal should be kept warm by means of rugs and bandages, and hot applications or stimulating liniments to the abdominal wall afford considerable relief. If there is any tendency to constipation, as may be the case when the inflammation is limited to the upper part of the small intestine, it is well to avoid the use of purgatives, which are rather apt to produce super-purgation. Relief should be afforded by the use of copious hot enemata and the administration of demulcents such as linseed tea, etc. If there is any danger of collapse, liq. strychninæ ʒii. or ether sulph. ʒss. may be given hypodermically. Strength must be maintained by suitable nourishment. In the acute stages it should be principally liquid, such as milk, oatmeal gruel, linseed mash, or hay tea. Later the appetite may be tempted with a little boiled oats, green meat, or steamed hay. These should be offered in small quantities and frequently; in fact most depends upon the actual nursing. In the parasitic form, by far the most useful agent is oleum chenopodii. During convalescence a course of alimentary and general tonics should be given, such as nux vomica, gentian, and alkalies.

Proctitis or Inflammation of the Rectum in Horses.—This form of enteritis may be due to specific causes, or may be brought about by mechanical causes. The latter are generally malicious in origin, as by a broom handle. A superficial form may also be due in the horse to the so-called seat-worm (false whip-worm, *Oxyuris cervula*). In mares, proctitis has also been caused by improper service, which has not infrequently brought about rupture of the rectum.

Symptoms.—In horses this gives rise to irritation of the lower bowel and of the anus, with frequent straining (tenesmus) and the passage of small quantities of fæces and obstinate rubbing of the tail. The latter also commonly occurs as the result of mange affecting the root of the tail.

Treatment.—When the rectum is ruptured treatment is not practicable. It has frequently been attempted, but so often disappointing in results as to be unjustifiable from an economic point of view. If the condition is due to a foreign body or hard fæces they should be removed manually, and sedative enemata administered, such as starch mucilage containing a little opium or belladonna. Sulphur sublimatum may be given by the mouth with the food. This acts both as a mild laxative and sedative to the rectum. The diet should be soft and demulcent. When the condition is due to seat-worms vermicidal enemata should be administered, such as infusion of quassia or strong brine. These should be given in small quantities of about Oi. at blood heat, and after the rectum has been previously emptied by back-raking. This may have to be repeated daily for a week or ten days.

Enteritis in Cattle.—Inflammation of the bowels in cattle is met with somewhat frequently. It may occur as a primary condition and is most frequently bacterial in origin. It also occurs in Johne's disease, tuberculosis, rinderpest, anthrax, septicæmia hæmorrhagica, malignant catarrh, etc. Enteritis may accompany gastritis due to nematode worms (parasitic gastro-enteritis). Coccidiosis of the bowel may also occur, setting up acute enteritis with dysentery.

Acute Enteritis in Cattle.—Although this affection may be and frequently is severe enough to produce fatal results, yet it is not so acute as enteritis in horses, generally running a more protracted course. It is also much less frequently caused by foreign bodies in cattle than in the horse. In cattle these agents are usually arrested either in the rumen or reticulum. The condition may be brought about by irritants in the foods, such as irritant chemical poisons as arsenic; irritant herbaceous materials, such as yew, rhododendron, croton seeds, and castor-oil seeds. The two latter may be met with in foreign-made feeding-cakes.

Symptoms.—The affected animal has a tendency to isolate itself from the rest of the herd, refuses food, ceases to ruminate. In milch cattle lactation is diminished. The mucous membranes are injected, and the temperature is elevated two or three degrees, occasionally more. The pulse is small in the early congestive stages, but may become larger later unless the condition becomes rapidly grave. There is evidence of some abdominal pain, with arching of the back, hollow flanks, and tucked-up abdomen. Tympany sometimes occurs, but is by no means a constant symptom. The bowels are at first slightly constipated, but this is followed by diarrhœa and tenesmus. In fatal cases the animal gets rapidly weaker, the fæces often blood-stained, the pulse smaller, and the temperature may before death become subnormal. In favourable cases after a day or two the patient becomes brighter, appetite and rumination gradually return, there is slight increase in milk, the diarrhœa is less copious, and the pulse improves in size and general tone, while the temperature gradually descends to the normal.

Treatment.—If the condition is known to be due to an irritant substance in the food it may be well first to administer a full dose of linseed oil together with the antidote, if known. If, however, it is bacterial in origin, and this is generally the case if there is much elevation of temperature, a purgative is contra-indicated. The old classical treatment for the condition consists of a combination of pulv. opii gr. xxx.-ʒi., catechu ʒii.-iv., and creta prep. ℥i. This both relieves the pain and reduces the scouring and meets with much success. It is probably improved by the addition of ammonium carbonate ʒii.-ʒiv. as a general stimulant, and should be given in cold gruel. This should be repeated daily until the diarrhœa ceases. Alternatively the pain may be reduced by other sedatives such as chlorodyne or chloral hydrate ʒss.-℥i. or potassium bromide ℥i. Internal antiseptics are also useful, such as salol ʒii. and tannoform ʒii., or hyposulphite of soda. When the acute stages are passed alimentary tonics, such as nux vomica ʒi., gentian and calumba āā ʒii., should be given twice daily. Attention should be paid to the feeding. If there is any suspicion that the food contains the irritant, there should be a complete change of diet, and while the patient is ill strength should be maintained by the careful administration of nutritive gruels. Water should be curtailed or withheld, and as the result of this the cattle will frequently drink the gruels and thus save forcible administration.

Proctitis in Cattle.—Inflammation of the rectum in cattle is extremely rare. It may occur, however, in the course of certain specific affections, such as rinderpest and septicæmia hæmorrhagica, and also in advanced cases of chronic bacterial enteritis (Johne's disease), to the articles on which the reader is referred.

Enteritis in the Sheep.—This condition occurs under very similar circumstances as enteritis in cattle. Acute enteritis is, however, much more serious in its effects and is more rapidly fatal.

The symptoms correspond closely with those of cattle, with the exception of the rapidity of development.

Treatment.—The same medicinal agents may be employed in corresponding doses. (The dose for sheep may in most instances be regarded as about one-fourth of the dose for cattle.)

Parasitic Enteritis in Ruminants (see p. 319).

Enteritis in Swine.—Inflammation of the bowels in swine is rare except in swine fever, tuberculosis, parasitic diseases, and in poisoning by irritant substances. The reader is referred to the articles on those affections.

Enteritis in the Dog and Cat.—*Catarrhal enteritis* is by far the most common form met with in these animals. It is most frequently specific in origin, and may occur as a complication of distemper, and of the so-called Stuttgart disease. Irritant poisons may also be responsible for producing inflammation of the bowels. In these cases it is usually complicated with gastritis. Catarrhal enteritis may also be produced by worms, particularly nematodes, the *Ascaris marginata* in the dog and *Ascaris mystax* in the cat. Tape worms can also set up the trouble, but they less frequently cause irritation of the bowels than the round worms. Enteritis in the dog may be chronic or acute. The chronic form is frequently diagnosed as indigestion, and may give rise to intermittent symptoms. The most serious feature about it is that with very slight provocation acute enteritis may supervene, running a very rapid course.

Symptoms.—Diarrhœa is the most pronounced symptom, which may be intermittent, or in acute cases continuous and very persistent. The animal is very depressed and has an anxious expression. The back is arched, and the abdomen tucked up, and the patient frequently objects to being taken up with the hand under the abdomen. There is frequently intermittent vomiting. The diarrhœa varies in character, the fæces sometimes being bright yellow, sometimes black, and at other times mixed with blood, and very offensive in odour. There is constant straining of the bowels or tenesmus, with the passage of only small quantities of fæces. This constant straining frequently leads the owner to the wrong conclusion that constipation or some obstruction exists. There is very marked loss of flesh, the patient becoming very thin and emaciated in a remarkably short time. The skin becomes hidebound, due to the lack of nutrition and the removal of subcutaneous fat, losing its elasticity, and if raised in a fold tends to remain in that position instead of rapidly resuming its normal condition. The temperature is elevated several degrees (105-106° F.), except in the advanced stages where collapse occurs. The temperature is then subnormal, the body surface, legs, and lips very cold, and the animal can scarcely stand. The course of the condition is very variable; in acute cases it may prove fatal within twenty-four hours, whereas in chronic cases it may last an indefinite period.

When enteritis is due to worms, a somewhat different train of symptoms is set up. The abdomen becomes distended and intermittent diarrhœa occurs. The temperature is usually normal. Vomition may occur, and the vomit frequently contains worms. Irritation of the skin may be present also, and somewhat distressing convulsions or screaming fits may be set up. This form is much the most commonly met with in puppies.

Treatment.—Nursing is of paramount importance in acute enteritis. Solid foods and water must be withheld. Nourishment must be afforded by means of white of egg with milk, to which a little brandy has been added. Albumen water, barley water, and concentrated meat juices may be offered, and if refused administered forcibly in small quantities. Various milk foods are of service, also Benger's food, cornflour, or arrowroot. Medicinally mild internal antiseptics and astringents should be given. Good results follow the use of chlorodyne mm. ii.-x. doses, which may be repeated three or four times a day. Tannoform gr. ii.-x., salol or salicylate of bismuth gr. ii.-x., quinine sulph. gr. i.-v. Enemata of normal saline solution (sodium chloride gr. iv. to water ℥i.) afford considerable relief. When the acute stages are passed great care must be taken with the resumption of solid foods. It is best even to avoid them altogether for some days, and then only to resume them in very small quantities. During this period a course of alimentary tonics, such as gentian and nux vomica, together with a little bismuth carbonate, may be given with advantage.

When the condition is due to worms, vermicides must be given, such as santonin gr. ss. for three- or four-months-old puppies, to gr. iv. for adults of the larger breeds, with a similar dose of calomel. Liquid extract of filix mas mm. v.-xxx. and oil of chenopodium mm. i.-v. are also useful, administered with a purgative such as castor oil. In a young puppy, syrup of iodide of iron mm. x.-xv. is the safest remedy, given in petroleum emulsion every morning, before the puppy has had any food. If the inflammation of the bowels is due to a known chemical irritant the antidote should be administered as soon as possible, while the effects and subsequent symptoms must be treated in the same way as acute enteritis.

Dysentery.—This is a condition in which blood is discharged with the fæces with or without diarrhœa. In reality dysentery is a symptom rather than a disease in itself. It may occur as the result of ulceration of the intestines or inflammation of the bowels. When there is a large amount of blood in the fæces it may result from a ruptured vessel in the intestinal wall, such as may have resulted from an ulcer, or even an injury by some sharp foreign body. If the origin of the blood is the stomach or the anterior part of the bowel, the fæces are usually black, owing to the decomposition of the blood before it is expelled. This form is called *melæna.* Dysentery is most commonly met with in coccidiosis and in specific diseases such as rinderpest and anthrax in cattle, purpura hæmorrhagica in the horse, swine fever in pigs, distemper and Stuttgart disease in dogs.

Treatment.—This must depend to some extent upon the disease of which it is a symptom. The dysentery itself may be controlled by the administration of chlorodyne or opiates or tannoform or iron salts, particularly the sulphate or sol. of perchloride of iron. Demulcents should also be given, such as linseed gruel or starch gruel to the larger animals, white of egg with milk or paraffinum liquidum for the smaller animals.

Diarrhœa in Young Stock.—This condition is a very serious one, and is due to a muco- or catarrhal enteritis. It is most commonly met with in calves and foals, and sometimes in lambs. There are two forms of the affection, one being infective and the other due to dietetic causes. The former condition is spread by contaminated foods, dirty udders, dirty suckling pails, and infective houses. The dietetic form may be due to sour milk and to improper artificial feeding. In foals it may result from allowing the foal to suckle the mare immediately she comes in from work. In calves it may be due to suckling cows with mastitis. A sudden change in the food of the mother is sometimes responsible for diarrhœa in the suckling, as when she may have been turned out into a new pasture of fresh clover. It may also occur at weaning time. (See also "White Scour," p. 77.)

Symptoms.—This form of diarrhœa is usually very offensive in odour, at first yellow and then greyish in colour, the latter particularly if the calf or foal be still suckling. The fæces stick about the tail, perineum, and thigh and often cause excoriation. The appetite is capricious, and there is a great falling off in condition. The temperature is elevated two or three degrees. As the condition progresses the extremities become very cold, and anæmia is set up with pendulous abdomen and hollow flanks. The eyes become sunken, and hidebound condition is set up and the temperature becomes subnormal. The infective form is much the more serious, and is likely to prove fatal in a large proportion of cases.

Treatment.—If the animal is still suckling, examine the mammary gland of the mother, and see if it is diseased or not. If it is, further suckling must be prevented and hand rearing substituted. If not suckling, special attention must be paid to the suckling pails, which must be scalded before and after use, and care must be taken to avoid contamination of any foodstuffs with infective excreta. The patient must be put into a warm, well-ventilated box and provided with clean bedding, which should be kept dry and frequently changed. The premises must be thoroughly disinfected. Medicinally it is frequently a good plan to commence with a dose of castor oil ℥i. or ℥ii., to remove indigestible materials from the alimentary tract, but this is a doubtful procedure if the infective form is suspected. Oxide or carbonate of magnesia gr. xv.-xx. together with ʒss.-ʒi. of chlorodyne to be given twice a day, or a little milk or starch gruel. Sodium hyposulphite destroys ferments, and is often found serviceable in doses of ʒii.-iv. twice daily. Pulv. rhei is also frequently given. It has the advantage of first causing purgation and being followed with an astringent after-effect. A very old and well-tried mixture for this condition consists of opium, catechu, and prepared chalk, which may be given twice a day in flour gruel in the following doses for calves and foals :

Pulv. opii gr. v.
Pulv. catechu ʒii.
Creta prep. ℥ss.

In the case of foals suckling working mares it is well to draw the milk from the udder when the dam comes in from work before allowing the foal to suckle. The diet of the dam must in each case be attended to, and she should be given a laxative easily digested diet, and a little sodium bicarb. ʒii.-iv. twice a day.

CONSTIPATION

Constipation in the Horse.—This is the arrest in the passage of fæces, and is generally caused by some defective secretions of the intestines or lack of muscular tone (atony) of the intestinal wall. It may also be due to a definite obstruction, such as calculus in horses or enlarged prostate in the dog. Constipation may be brought about by dry innutritious foods, an insufficient water supply, and lack of exercise. It may also occur as one of the symptoms in the early stages of fevers owing to the temporary reduction of secretions. Constipation is more commonly met with in old than young animals, and is most frequent in dogs, cats, and pigs.

Symptoms.—The essential symptom is the retention of fæces or the passage of fæces in smaller quantities than normal. In the latter case they are harder in consistence than normal, usually dark in colour and mucus-coated, occasionally offensive in odour, particularly if the condition is due to torpidity of the liver. The appetite is capricious and the horse is somewhat depressed and does not perform his work with his usual vigour. The mucous membranes are more or less dirty-looking and may be slightly yellowish. The urine is darker in colour than normal, and precipitates a quantity of yellowish deposit on standing. The pulse is usually soft and sluggish and lacking in tone. If the constipation lasts twelve or twenty-four hours colic is likely to supervene.

Treatment.—This depends somewhat on the cause. It is usually advisable to avoid violent purgatives. A mild laxative may be given, such as a small dose of linseed oil (Oi.-i.ss.) together with a laxative diet consisting of linseed mash or bran mash or green foods. Long hay should be withheld for the time. As an alternative to the oil, calomel ʒi. or aloes ʒiv.-ʒv. or sulphate of magnesia ℥vi.-℥viii. may be given. These should be followed subsequently by a course of alimentary tonics to improve the general tone and secretions. The best consist of nux vomica and gentian, together with bicarbonate of soda and the addition of sulphate of soda, if there is any tendency to jaundice. The administration of copious hot enemata materially assists in affording relief, and a little walking exercise should be given. In order to prevent the condition recurring it is often advisable to add about a wineglassful of linseed oil to the morning feed every day for a considerable period.

Constipation in Cattle, Sheep, and Goats.—This is by no means infrequent, and may be due to similar causes as in the horse. In some parts of England a bovine affected with constipation is said to be "staked."

Symptoms.—In the first place, there is an absence of fæces or the passage of small quantities of firm, hard, mucus-coated fæces. The animal feeds spasmodically and ruminates in a similar manner. In milch cattle the milk yield is reduced. Grunting and grinding of the teeth are very common symptoms. There is usually some abdominal pain and paddling of the hind feet. Great depression and indisposition to move about. If the animal is at pasture it tends to separate itself from the rest of the herd. There is usually no alteration of temperature or respiration.

Treatment.—This has usually to be a little more vigorous in cattle than in the case of horses. Except in obstinate cases, however, violent purgatives should be avoided, since they are frequently followed by astringent after-effects, which may result in a recurrence of the constipation. The best agents to use for cattle are linseed oil Oiss., to which a little turpentine ℥i. may have been added with advantage. Sulphate of magnesia ℥viii.-xii. in a quart of water is also good. This may be followed by alimentary tonics, such as nux vomica ʒii. and carb. of amm. ʒiv. twice a day. The laxative should be repeated daily for two or three days unless free operation of the bowels is obtained. For sheep and goats the same agents may be used in about one-fourth the doses for cattle. Enemata of soapy solution also afford relief. Attention must be paid to the diet, which should be of a laxative nature, coarse rough herbage being avoided. Linseed cake is very useful, but cotton cakes are better withheld.

Constipation in Pigs.—This may be brought about in pigs through overfeeding with dry grain and meals, particularly with an insufficient amount of exercise and defective water supply. It may also be due to feeding on acorns and sometimes from eating hair or sand. It is a common complication of such specific affections as swine fever and tuberculosis.

Symptoms.—At first there is a passage of fæces in small quantities, firm and dark in colour, and later a cessation of defæcation. There is usually loss of appetite and excessive grunting and straining, and considerable depression. The temperature is not affected. The skin may become discoloured, and the pig has a tendency to bury himself in his bedding.

Treatment.—If the pig is taking any food, opening medicines may be given mixed with it, such as castor oil ℥ii.-iv., sulphate of magnesia or sulphate of soda ℥ii.-iv. These may be repeated the next day if they are not effective. If the pig is not feeding, these agents may be administered by drenching; or, better still, a combination of jalap ℥ss.-i. with calomel grs. v.-grs. xx. may be administered in the form of electuary, being placed in the mouth by a flat piece of stick. Attention should be paid to the food. Further access to acorns or coarse herbage should be prevented, and where possible butter-milk or whey should be supplied, together with an allowance of succulent green foods. Copious hot enemata of soap solution may also relieve the condition, and the pig should be compelled to take exercise.

Constipation in the Dog.—This condition may be simply one of partial arrest in the passage of fæces or may be much more serious, a condition of impaction of the colon. The former condition is predisposed to by too constant feeding on starchy foods, by the presence of an enlarged prostate, or by inflammation of the anal glands. It may also occur as a symptom of any simple fever, and is usually present in cases of uncomplicated gastritis. The more severe condition of impaction of the colon occurs most commonly in old dogs, and is predisposed to by laziness and sedentary habits.

Symptoms.—There is diminished frequency of defæcation, the fæces being hard and firm and expelled in small pieces. Later no fæces are passed, the dog becomes very dull and lies about in a depressed condition. He may eat fairly well for a day or two and then refuse food. Vomiting may also occur, and in some obstinate cases the vomit may consist of fæcal matter which has been returned to the stomach from the bowels by a process of backward peristalsis. On palpation through the abdominal wall the hard fæces may be felt in the intestines, sometimes in considerable quantities. In obstinate constipation in dogs it is quite common for weakness of the hind limbs to occur, which is followed by progressive paralysis (paraplegia). Even in this condition the animal retains full consciousness and is sometimes surprisingly bright, though he may be quite unable to support himself on his hind legs. He may pull himself about, dragging his hind legs behind him. This paralysis passes away when the condition of obstinate constipation is removed.

Treatment.—The arrested fæces should first be removed by means of hot enemata frequently repeated. It may be necessary also to insert small whelping forceps in order to grasp and break down some of the hard masses. This procedure should be persisted with until practically all the material has been removed, which can be ascertained by palpation through the abdominal wall. If this is properly effected there is no necessity to administer a purgative, but if it is found impossible to remove the whole of the fæcal mass a purgative should be administered. In such cases the combination of castor oil with syrupus rhamni (ʒii. to ℥i. of each according to the size of the dog) is probably the best. Calomel gr. i.-iv. is also very useful. After relief has been afforded a course of nerve and alimentary tonics should be administered, such as tincture of nux vomica m. v., tinct. of gentian and tinct. of calumba āā m. x., together with aromatic spirits of ammonia m. xv. This should be given twice daily after food. A course of petroleum emulsion or paraffinum liquidum prevents a recurrence of the condition. For a similar purpose carbonate of magnesia gr. v. and sulphate of magnesia gr. xv. once or twice daily gives excellent results. Regular exercise should be enforced, and attention paid to the feeding. Bones and biscuits should be avoided. The best foods consist of tripe or paunch, horseflesh, and raw or partially cooked liver. Where paralysis has occurred a stimulant may be applied to the loins either in the form of a mild blister or a stimulating liniment. The administration of nux vomica and arsenic is particularly indicated in these cases.

Constipation in the Cat.—This condition is most frequently met with in cats leading sedentary lives, such as castrated males or spayed females, and is largely due to laziness and insufficient exercise. It may be due to the taking in of considerable quantities of fur by licking. Constipation may also occur as a symptom of any febrile condition.

Symptoms.—Dulness and depression are the chief signs of constipation. The cat refuses food and milk, and growls on being interfered with. No fæces are passed, but on manipulation of the abdomen the arrested ingesta can be felt in the intestines, usually the colon.

Treatment.—This is quite simple, and consists in the administration of gr. i. calomel pill, or castor oil and syrupus rhamni āā ʒii. A hot enema may cause the expulsion of the hard fæces. After-treatment consists in attention to diet and insistence upon exercise.

Proctitis and Impaction of the Rectum in the Dog.—Inflammation of the rectum is more common in the dog than in any of the other domesticated animals. It may arise as the result of a collection of hard dry fæces or splinters of bone, or sometimes a rather large piece of bone which may have successfully passed along the rest of the alimentary tract only to become arrested in this situation. This accumulation is often predisposed to by a condition of inflammation of the anal glands which in itself renders defæcation painful. Proctitis frequently occurs in the intestinal form of distemper.

Symptoms.—When the rectum is impacted with bones there is usually considerable pain during attempts to defæcate, so that a dog often refrains from evacuating his bowels until he is absolutely compelled to do so, thus aggravating the condition by leading to further accumulation. In the case where a piece of bone becomes fixed across the rectum, just inside the anal rim, the dog frequently screams when attempting to defæcate, and may bolt or run away in a state of frenzy. Such cases have often been mistaken for rabies. When the obstruction is removed, and also in catarrhal proctitis of distemper, there is more or less constant straining or tenesmus. When this occurs, owing to the fact that little or no fæces are passed at the same time, owners frequently form the opinion that the dog is constipated, whereas it so often happens that the bowel is completely emptied. Diagnosis is confirmed by digital exploration of the rectum.

Treatment.—The rectum should first be explored, and if there is any accumulation of fæces the latter should be removed digitally, with the assistance of repeated enemata either of soapy water or oil or glycerine and water. If this fails the mass should be removed by means of small whelping forceps. Subsequently repeated injections of some sedative or demulcent is advisable, such as starch enema, to which may be added a little tincture of opium; enemata of normal saline solution also afford relief. If the anal glands are impacted they must be squeezed out. Mild laxatives and alimentary sedatives should be given by the mouth, amongst the best being paraffinum liquidum or petroleum emulsion, to which a little chlorodyne mm. ii.-x. may be added. The diet for a while should consist of meat or paunch or milk foods; bones and biscuits to be avoided.

Inflammation of the Anal Glands in the Dog and Cat; Inflammation of the Cloacal Glands; Impaction of the Anal Glands; Pruritus Ani.—On either side of the anus in the dog and cat there is a group of glands leading into a common cavity with a small duct which opens just at the margin of the anus. Its function would appear to be to secrete material which is squeezed out during defæcation of firm fæces, the passage of which it facilitates by lubricating the anal orifice. The duct of this gland not infrequently becomes choked, either by the inspissation of some of the secretion or by a small amount of fæces, or in some instances by segments of tape worms. The secretion is thus imprisoned and the gland becomes distended and inflamed, causing a painful condition varying in degree in proportion to the distension. This condition undoubtedly occurs more frequently in town-bred and pampered dogs, and is apparently aggravated by the artificial methods of feeding whereby the fæces are in a chronic condition of softness. As the result of this the glands are not evacuated as they should be during defæcation, and blockage of the duct ensues. It is much less frequently seen in cats than in dogs.

Symptoms.—Frequently the first indication of the condition is the animal squatting down and sliding along the carpet or pavement, with the obvious intention of rubbing an irritated anus. He also frequently licks the part, and in the case of playful dogs, such as pugs, he runs round and round very rapidly after his tail, a circumstance which is often interpreted by the owner as an indication of playfulness. The affected animal also has a tendency to get under chairs and rub his croup with evident satisfaction, and sometimes this is carried on so persistently as actually to cause lesions of the skin over the sacrum, leading to a diagnosis of eczema. The irritation of the croup is apparently reflex from the irritated anus. In a number of cases the impacted gland may become so distended as to cause very great distress and weakness of the hind limbs with a rolling gait and sometimes reflex vomiting. In other cases the distended gland bursts outwardly, thus setting up a fistula of the anal gland. The painful nature of the condition often leads to the dog refraining from passing his fæces, and to the production of severe impaction.

Treatment.—Attempts to relieve the glands should first be made by squeezing them. The finger and the thumb should be pressed on the outer side of the glands forward into the perineum in order to exert pressure in the backward direction. In most cases this is successful, and if the material of the gland is very liquid it may be forced out in a jet a distance of some feet. It may sometimes be observed that the first spot that is expelled actually consists of a discoloured segment of tape worm. At other times the contents are thicker, while occasionally they are semi-solid. It may be necessary to squeeze each gland individually, in which case the forefinger is inserted into the rectum. Pressure may fail to relieve the condition, and it is then necessary to pass either a probe or, better still, a very small grooved director down the duct of the gland, to remove the obstruction, a procedure which requires a certain amount of deftness and experience to accomplish with ease. After evacuating the glands hot fomentations should be applied, and the irritation may be still further reduced by the application of a sedative astringent ointment. A useful application consists of pulv. aluminis, pulv. opii āā gr. x., ext. hamamelidis ʒi., adipis ℥i. A little of this should be well rubbed into the anal rim twice daily. If these methods fail to relieve the condition, surgical measures must be resorted to; either the entire gland must be dissected out or a grooved director may be passed down the duct, and by means of a sharp scalpel the duct and the cavity of the gland should be exposed. Care must be taken to avoid wounding the rectum and causing a fistula. When the condition is associated with tape worm, tæniafuges such as freshly ground areca nut or its ethereal extract (tenaline) should be given. No other medicinal agents are required by the mouth, beyond mild laxatives, and the bowels should be kept in a semi-relaxed condition by means of a suitable diet. Bones should be avoided for some time, and also any food containing condiments.

Hæmorrhoids; Piles.—This condition in dogs is exceedingly rare. In the course of very extensive experience in dog, the writer has only encountered the condition once. Inflammation of the anal glands is extremely frequently mistaken for hæmorrhoids in the dog, and particularly so by the layman. True hæmorrhoids consist of dilated veins of the anus and rectum which protrude during and after defæcation and are sometimes hæmorrhagic. Relief may sometimes be afforded by attention to the bowels, and the local application of ointment recommended for inflammation of the anal glands. If this fails surgical treatment is necessary.

COLIC

By the term *colic* at the present time is meant a collection of symptoms indicating abdominal pain, usually acute and suddenly occurring. In reality, however, the term refers to spasm of that portion of the large intestines designated *the colon*. The majority of cases of colic in the horse are probably due to some affection of this portion of the intestine. By general consent, however, the term has gradually been extended to include almost any abdominal affection associated with pain. It is therefore obvious that the term colic should not be regarded as a disease of itself, but is only an indication of one of many affections of the abdominal cavity whose only common feature is the production of more or less intense pain. When the pain is very intense it is called *acute* colic. When the pain is dull and less violent it is called *subacute* colic. Colic is sometimes termed " true colic " when associated with spasm of the colon, and " false colic " when due to any other causes. This distinction, however, is now falling into disuse. The horse is by far the most common subject of colic amongst the domesticated animals.

Colic in the Horse ; " Gripes " ; " Belly-Ache "

Causes of Colic.—In the horse there are several conditions or circumstances which predispose to the production of abdominal pain. The relatively small size of his stomach, together with its peculiar shape and disposition, and the necessity for frequency of feeding, predispose to derangement of that organ, with resulting pain and colic. The peculiarity of its arrangement concerns both the entrance of the œsophagus into the stomach, which is so arranged as to prevent under ordinary circumstances the return of objectionable or irritant materials by vomiting, and also the exit from the stomach through the pylorus and duodenum. This latter consists of an S curve which may easily be occluded by pressure if the stomach should become over-distended either by food or gas. This course prevents the passage of the food or gas from the stomach into the intestines, and so intensifies the condition of impaction or tympany. Another peculiarity in the horse's intestines is the presence of the numerous folds or puckers (*valvuli conniventes*), in which food material may become arrested in any degree of stasis and thus predispose to fermentation and flatulence. The great susceptibility of the horse to vicious parasites which frequently wound the mucous membrane, and in some instances pass a portion of their life-history within the bowel wall, is a predisposing cause to abdominal pain. Moreover, the mucous membrane of the intestines of the horse appears to be peculiarly sensitive to irritation. Heredity has been suggested as a predisposing cause to colic, and this is probably open to dispute. There is a possibility, however, of the transmission of a more than usually sensitive nature or character of the intestinal mucous membrane in certain cases, and if that be so, then one must admit the possibility of hereditary predisposition to colic. Age must also be included as a predisposing cause to colic. Young horses are more frequently affected with colic due to parasites than adult horses, while aged horses are more prone to colic due to indigestion resulting from improper mastication owing to irregular teeth, or from debility of tissue, causing reduced secretions and peristalsis.

Immediate or Exciting Causes of Colic. Food and Feeding.—As already stated, owing to the relatively small size of a horse's stomach he should be fed in small quantities and frequently. Neglect of this precaution is apt to cause overdistension of the stomach by hungry horses, while a similar effect is produced by greedy feeding. In each case the condition is intensified by improper mastication. Irregularity in the times of feeding is, however, the most frequent cause of colic in horses, and this is especially the case if a horse has been hard at work beyond his usual feeding time and has arrived in the stable in a more or less exhausted condition. The character of the food must also be taken into account. Improper foods such as those containing undue proportions of indigestible fibre, such as oat chaff and chopped straw, or foods of inferior quality or damaged foods, are likely to cause abdominal pain, either by the irritant effects causing inflammation of the bowels or by setting up impaction of the bowels or by causing excessive fermentation and tympany. A sudden change of diet may have a similar effect. It is therefore advisable that when a change of diet is necessary the change should be brought about gradually. Other foods likely to set up colic are those which easily ferment and those which are likely to swell after being taken into the stomach, such as wheat and barley. A horse should never have a full meal immediately before being put to work, and this is especially the case with horses doing fast work. Boiled foods sometimes produce colic, and that is probably due to greedy feeding and over-distension of the stomach. If colic occurs in a stud due to improper food it is usual for several to be affected at the same time.

Water.—It is said by many that giving horses cold water directly on coming in from work is a common cause of colic. Experience in some of the largest studs, however, undoubtedly negatives this view, except in those cases where the horse is brought in very exhausted and thirsty, and then allowed to take excessive quantities of cold water. When a horse comes in exhausted he should not be allowed a large draught of water; it is better to provide him with half a pail of warm oatmeal gruel, which acts as a stimulant and restorative, and temporarily assuages his thirst. He may be allowed water to drink half an hour or so later, when he is somewhat rested. In some of the big omnibus stables in London, when it was the custom to withhold water from the horses on coming in from a journey, cases of colic were very numerous, but when the practice was changed and the horses were turned loose to the water trough and allowed to drink as much as they wanted, the number of cases of colic very rapidly diminished. It is probable that more colic is caused by withholding water than by allowing it at such times. It is always a good plan also to water horses before feeding. If the converse order is practised, undigested food is apt to be carried through the stomach into the intestines, and so set up indigestion and colic. Except in very flagrant mismanagement of its supply, water can therefore be disregarded as, at any rate, a prolific cause of colic. Water contaminated by *lead* may cause colic, especially a soft water stored in leaden cisterns or conveyed in leaden pipes, particularly if the latter are new or have been in disuse for some time. The exciting cause of colic in this case, however, is lead and not water.

Colic may also be caused by various *obstructions*, such as stricture of the pylorus or stricture of the intestines, calculi, or tumours either within the bowel itself or pressing on the intestine from the outside. A common cause is impaction of the bowels, particularly the large colon, which may be due either to defective secretion of the intestinal glands or defective peristalsis (atony or paralysis of the bowel). Other exciting causes include intussusception or telescoping of the bowel, volvulus or twist of the bowel, and inflammation of the bowels. The presence of parasites in the lumen of the bowel may set up more or less acute abdominal pain, but they are a more prolific cause of pain when they are passing the immature stage of their life-history in the substance of the bowel wall, thus producing a deep-seated enteritis, as in the case of some of the sclerostomes. Another form of verminous colic is due to the presence of immature parasites in the large mesenteric blood-vessels setting up aneurism, as in the case of *Sclerostomum vulgare* in colts. Retention of the urine is another cause of colic, and this may be due either to spasm of the neck of the bladder or to pressure on the neck of the bladder from without.

Acute abdominal pain arising from diseases of the kidney is known as *renal* colic, and when arising from diseases of the liver it is called *hepatic* colic.

The *pain* in colic may be due to several causes. It may be due to direct irritation of the nerve-endings in the delicate mucous membrane by the irritant, or it may be due to irritant toxins, as in the case of specific enteritis and anthrax. It may also be due to stretching of the walls in the intestines and the nerve-endings, as in tympany, or to compression of the nerve-terminals during spasm of the intestinal muscle. In other cases the pain is due to interference of the circulation of the part, as in strangulation and aneurism.

The *cause of death* in colic cannot always be clearly explained. In acute cases, where death may occur within twelve hours or so, it is probable that shock and acute nervous exhaustion may account for it. In other cases it is undoubtedly due to the absorption of toxins produced by bacteria having been enabled to penetrate the intestinal wall or peritoneum, either as a primary cause of the colic or due to reduced vitality of the tissues brought about by the primary cause. In a few cases rupture of the stomach or of the bowel accounts for death.

For convenience of description colic may be divided into *spasmodic colic* and *flatulent colic*. The latter includes those cases in which fermentation and the accumulation of gas are a predominating feature. All other forms are included in the term spasmodic colic.

Spasmodic Colic.—Under this heading are included forms of colic due to a variety of causes which will merit individual treatment in succeeding articles. It is convenient, however, to consider together a number of symptoms which are more or less common to them all. A veterinary surgeon, however, should not be content with making a diagnosis of spasmodic colic, but should proceed further in his examination of the patient, with a view to ascertaining, if possible, the actual cause of the spasm.

Symptoms of Spasmodic Colic.—The symptoms usually occur very suddenly in the form of spasms more or less acutely painful, but often intermittent in character. The duration of the spasms and also the intervals vary considerably. The horse breaks out in perspiration, often in patches, and has a very anxious expression of countenance. The respiration is blowing and accelerated. The pulse is hard and accelerated, and otherwise varies in character according to the origin of the colic. In those cases associated with congestion of the bowels or internal organs the pulse is small, while in many other cases it is unaltered in volume. The mucous membranes are injected, the temperature in the early stages is normal, but later may become raised owing to excitement, and probably also to absorption of toxins. There are usually frequent attempts at defæcation, fæces being passed in small quantities only, and in obstructive cases defæcation soon ceases. In other cases where there is no obstruction the fæces become soft, with a tendency to diarrhœa. The patient also frequently stretches as if to urinate, often in vain owing to spasm of the neck of the bladder. In such cases if micturition takes place it is often a favourable sign, since it indicates some relaxation of the spasms. The patient frequently looks round at his side, kicks forward at the abdomen, and lies down, rising again at short intervals. When down he may roll about with considerable violence, often rising again immediately after rolling.

Treatment of Spasmodic Colic.—There are two main objects in view in the treatment of spasmodic colic, namely, the relief of pain and the removal of the cause. Obviously relief of pain alone will not be effective if the cause is allowed to remain in operation, hence an attempt to arrive at a proper diagnosis is of paramount importance in the treatment of colic. Since colic may be associated either with an obstruction in the bowels or with irritation and diarrhœa, one should avoid the use of any agents which may affect the action of the bowels in either direction until a definite diagnosis is arrived at. The choice of an anodyne, therefore, should be directed to the selection of one which will reduce the pain through the higher nervous system without any direct action upon the alimentary tract. Opium and morphia are very excellent anodynes, but as they have a very pronounced effect in reducing activity of the bowel walls, and also secretions, they should, in the first instance at any rate, be avoided. Probably the best anti-spasmodic to commence with is chloral hydrate ℥i.-i.ss. administered in water in the form of a drench. Another useful combination consists of oleum terebinthinæ ℥ii., ether meth. ʒi., ol. lini Oi. Cannabis indica ℥ss.-i. is also useful, and has an additional advantage of being more durable in its effects, which may last twenty-four hours or so. If there is no obstruction or constipation, then tinct. opii ℥ii.-iv. or pulv. opii ʒi.-ii. may be given, the former in draught along with ether meth. ʒi., or the pulv. opii may be given in bolus. In many cases, if the horse is at all violent it is impossible or undesirable to attempt to drench him. Recourse may then be had to an injection of morphia hypodermically in fairly large doses, from gr. v. to viii., or to a combination of morphia gr. iv. with atropine gr. i. The latter combination is probably less objectionable in cases of constipation than morphia alone, but is not recommended if it is possible to administer chloral hydrate or other anti-spasmodics not affecting the bowels. In colic complicated with constipation relief should also be afforded by the administration of a dose of physic, such as aloes ʒiv.-vi. in bolus or, if more rapid result is desired, eserine gr. i.-ii. hypodermically, or eserine combined with pilocarpine in half that dose. Arecoline in a similar dose is very useful. In uncomplicated cases of constipation these alkaloids will frequently produce repeated evacuation of the bowels in from ten to twenty minutes. Barium chloride may also be used for the same purpose in doses of gr. x.-xv. injected intravenously or ʒi.-ii. given by the mouth. Barium salts, however, are not to be recommended if the former alkaloids are available, since they produce a simultaneous contraction of the bowels, and not a wave of contraction as in the case of eserine, etc. Consequently barium is more likely to result in rupture of the bowel than those agents.

The patient should be backraked and enemata should be administered. Copious hot enemata of soap solution stimulate peristalsis and relieve abdominal spasms. On the other hand, lukewarm enemata are likely to be retained and assist to soften the fæces in cases of constipation. The use of the stomach tube may also be indicated (see p. 305). If the bladder is distended it must be relieved by the passage of the catheter, which must be thoroughly cleansed and disinfected before being used. The application of hot cloths to the abdominal wall affords very considerable relief. For this purpose a blanket should be wrung out in boiling water and applied as quickly as possible to the abdominal wall, and then covered with a waterproof sheet. If this is done it will remain hot for several hours, and reduce the necessity of frequent repetition. As an alternative the application of a mustard plaster may be resorted to, but if this is used it must be applied cautiously, otherwise it is apt to cause great swelling and even sloughing of the skin. In the case of thin-skinned horses it is better avoided or washed off after about ten minutes or so. In the case of the thick-skinned horses it may be left on if it has not been applied too vigorously.

Flatulent Colic; Intestinal Tympany; Windy Colic.—This form of colic is due to the presence of gases in considerable quantities in the bowels, particularly the cæcum and the large colon. It is often due to the administration of foods of a succulent nature, particularly if taken in unusual quantities by animals that are not accustomed to them. Animals accustomed to take similar foods may do so with impunity. The foods likely to cause the condition include fresh green clovers and vetches, boiled foods, as wheat and barley, wet brewers' or distillers' grains, new oats, oat chaff, and uncooked potatoes. Roots of any kind may set up the condition if the animal is not accustomed to them. Damaged and mouldy foods are also fairly common causes of intestinal tympany. Any condition which involves the arrest of food predisposes to fermentation and to tympany. Such predisposing causes include defective intestinal secretions and defective muscular power of the intestinal wall. Tympany may also occur in the course of certain well-defined diseases of the intestine—volvulus or twist, intussusception, and calculus. "Wind-sucking" may also cause tympany, first of the stomach and, if continued, of the intestines.

Symptoms.—Flatulent colic usually develops rather suddenly and causes a varying degree of abdominal pain which is more or less continuous. In mild cases the pain is subacute, but in bad cases it may become very acute and distressing. The horse walks round and round the box, kicks forward at the abdomen, frequently looks round at his sides, and breaks out into patchy sweats. The abdomen becomes somewhat distended with gases. This is observed in the flanks, particularly on the right side, and percussion reveals marked resonance. The mucous membranes are injected, while the pulse is quick, large and hard, but in very advanced stages may become quite small. Respiration is laboured owing to the pressure on the diaphragm by the distended bowels. The horse frequently crouches as though to lie down, but only actually lies down in subacute cases, and even then quickly rises again. In acute cases, although he may crouch low, he rarely reaches the prone position, since any extra weight on the abdominal wall as would be produced by lying down only serves to intensify the pain. In very advanced stages, however, the patient is quite unable to stand, he staggers and may actually fall. This is very dangerous, and may lead to rupture of the bowel. There is usually frequent passage of fæces in small quantities, sometimes accompanied by flatus, which may afford some relief. Pressure on the bladder by the distended bowel causes frequent attempts at micturition, often in vain. Rectal exploration enables one to feel the bowel distended with the gases, which is usually pressed well back into the pelvis. Compared with spasmodic colic the patient is as a rule less violent, but the symptoms are more continuous. In fatal cases death may be due either to asphyxia from paralysis of the diaphragm caused by the pressure of the distended bowels, or to rupture of the bowel or rupture of the diaphragm.

Treatment.—The first principle consists in the removal of the cause either by preventing further production of gases or by the direct removal of the gases. The former may be obtained by the administration of antizymotics and general stimulants. Amongst the best for this purpose is oleum terebinthinæ ℥ii., spts. ammon. aromat. ℥i.-ii. with ol. lini Oi.ss., Ft. Hst. Another useful combination consists of ol. terebinthinæ ℥ii., ether meth. ℥i., aloes ʒi., ol. lini Oi.ss. Chloral hydrate ℥i. in water Oi. is also very useful, being simultaneously antizymotic and antispasmodic like the previous combinations. It is often advisable also to administer a dose of physic such as aloes, unless there has been a repeated administration of oils. The drastic purgatives, such as eserine, pilocarpine, and arecoline, are contra-indicated if the tympany is at all severe, as they may lead to rupture of the bowel. The administration of opium or morphia or chlorodyne is not to be recommended if the former agents are available, as they still further reduce the secretions and peristalsis which may be the original cause of the condition. If the tympany is very excessive it should be relieved by means of the trocar and the cannula, with which the distended colon or cæcum may be punctured. The instrument should be 9 or 10 inches long with a comparatively small bore, not exceeding ¼ inch. It should be boiled before being used. The seat of puncture is high up on the right flank, at the most prominent point between the last rib and the angle of the haunch. The instrument should be inserted downwards and forwards after disinfection of the skin, and as the trocar is withdrawn the gas should escape through the cannula. The gases are rarely offensive in odour, and consist principally of marsh gas and carbonic acid gas. Occasionally a little sulphuretted hydrogen is present, rendering it offensive. If this fails to remove the gas the trocar must be reinserted and withdrawn with the cannula, to be reinserted lower down on the same side. It is very rare that more than one puncture is necessary, but in cases of necessity one should not hesitate to puncture several times until relief has been afforded. This procedure may appear to be dangerous, but failure to relieve the distension is no doubt still more dangerous and likely to lead to fatal results. Occasionally it may be necessary to puncture on the left side of the abdomen if distension of the abdomen is more pronounced on that side. Some practitioners recommend the puncture of the distended bowel per rectum, but one fails to see any advantage in this method. When the gas has escaped, concentrated disinfectants may be injected through the cannula directly into the bowel. In this way they arrive directly at the seat of fermentation, and produce their effect more rapidly than if administered by the mouth. It is sometimes recommended that the cannula should be left in to allow of the escape of any gases produced. This is rarely necessary, but if it is practised it must be secured in position either by sutures to the skin or by a tape round the horse's body. To remove the cannula, the trocar must always be first reinserted in order to remove any ingesta which may have become lodged into the mouth of the instrument, and which on withdrawal might fall into the peritoneum and set up peritonitis, or become lodged between the muscles or under the skin and cause abscess formation. Enemata of hot water stimulate peristalsis and may stimulate the expulsion of gases. They should be repeated frequently during the attack. Lavage may also be found serviceable (see p. 305). Hot applications to the abdominal wall afford some relief, or alternatively a mild application of mustard, which should be washed off in ten or fifteen minutes, especially in the case of a thin-skinned horse. After relief has been afforded the patient must be kept under observation for fear of a recurrence. Subsequent treatment consists of ensuring several days' rest for the patient on a nutritious and not too bulky diet. A course of alimentary tonics should also be prescribed, consisting of gentian, nux vomica, and calumba.

Colic due to Special Causes

Intestinal Obstruction ; Calculus or Enterolith.—This is the most common cause of fatal obstructive colic in the horse. Calculi in horses consist of remarkable concretions made up either of mineral salts which have been taken in with the food or of organic matter, mainly animal or vegetable hair or fibre. These substances become deposited around a central nucleus which may consist of a small piece of metal, as a nail or button, or a bit of stone, gravel, or leather. Occasionally the nucleus consists of a parasite, and sometimes of oat husks. Certain foods rich in salts are regarded as predisposing to the formation of calculi, and this is particularly the case with bran, which is rich in mineral salts. This is supported by the assertion that millers' horses are said to be most frequently affected. In any case it is the foreign body which is the principal factor. The frequency with which nails are found amongst corn has justified the erection of large plants with magnets for the removal of such like bodies. In big studs where this has been carried out the incidence of calculi has been enormously reduced or entirely eliminated. Calculi probably commence in the majority of cases by the foreign body being arrested in one of the folds of the large intestines or in one of the flexures of the large colon. The increase in size and weight is probably slow, in which case the calculus remains freely movable in the lumen of the intestine. On the other hand, the increase of weight is sometimes very rapid, and the calculus appears to make for itself a sac or pouch in the intestinal wall in which it develops. It may in this way reach very large dimensions without causing any apparent inconvenience, and may actually attain the weight of 50 or 60 lbs. without having been suspected during life. The size which causes the most trouble is that slightly larger than a cricket ball, which can move about freely in the large colon, and eventually becomes arrested at one of the flexures or becomes wedged into the entrance of the floating colon, causing a fatal obstruction.

Calculi vary in shape ; the majority are spherical, some are ovoid and others pyramidal, showing facets produced by several calculi rubbing together. There may thus be a single calculus which may attain a large size, or they may be multiple of varying sizes, sometimes faceted, sometimes showing no indication of friction with others. The weight of the calculi also varies, those composed mainly of mineral salts being much heavier than those composed largely of organic matter.

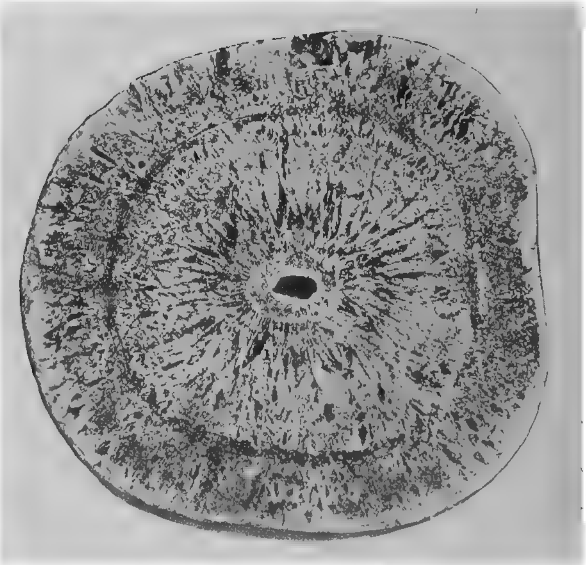

Fig. 72.—Intestinal calculus of horse sawn through to show metallic nucleus ($\frac{2}{3}$ actual size).

Composition of Calculi.—The heavy mineral calculi, which are sometimes called *true calculi*, consist chiefly of phosphates and carbonates of calcium, magnesium, ammonium, and silicon, held together by mucus and other organic matter. These calculi appear to be deposited in concentric layers which can be more or less easily chipped off. When they are sawn through the middle, the nucleus is generally found in the centre and the body of the calculus shows both radiating and concentric lines. The surface of mineral calculi is usually smooth, occasionally crystalline.

Organic calculi are composed chiefly of oat husks, oat hair, barley awns, dust, and sometimes animal hair held together by mucus, etc. These are sometimes called *false calculi.* They are very light in weight compared with mineral calculi of the same size, and are mostly globular in shape. The majority have an irregular bosselated surface, and are sometimes erroneously called mulberry calculi. The latter term is better limited to those cases where a large calculus appears to be made up of the union of a number of smaller ones.

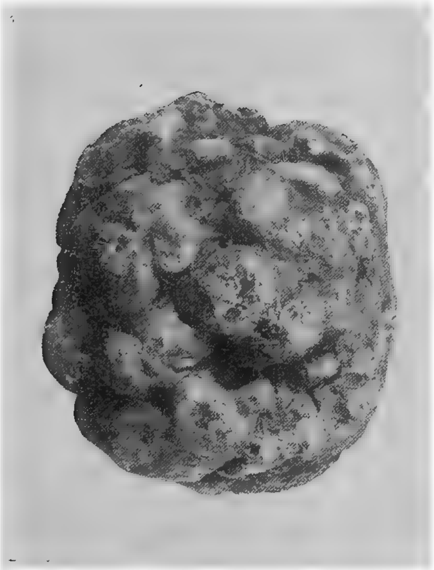

FIG. 73.—Bosselated intestinal calculus from horse ($\frac{3}{5}$ actual size).

Symptoms of Calculus.—The symptoms are those of recurrent spasmodic colic. The attacks may come on rapidly and vary in duration. They may give rise to obstruction of the colon, and when this occurs at one of the flexures it may be relieved, the symptoms passing away after a varying period. If, however, the calculus is arrested at the commencement of the floating colon, a complete obstruction may occur and prove fatal in from four to seven days. In other cases, if the calculus is not quite so large it may be passed through the small colon, and out with the fæces. As in the case of obstruction from other causes, there is a cessation of the passage of fæces for a varying period, and the severity of the spasms may be subacute or acute. It is not an uncommon thing for numerous smaller calculi to be passed without any symptoms having been caused. The definite diagnosis is difficult, and one has to rely largely upon the history of the case, which may consist of repeated attacks of colic from time to time. The manner of feeding, particularly if the food has contained much bran, may assist one in arriving at a diagnosis, and suspicion may also be aroused if the horse has been fed on foreign corn. Enemata which may have been administered in treatment are rarely retained, and this also affords suspicion. Positive diagnosis, however, is impossible unless the calculus can actually be felt per rectum.

Treatment.—Since the only positive diagnosis is that of spasmodic colic with obstruction, the treatment is the same as for spasmodic colic. In rare cases, however, the calculus may be felt per rectum and removed manually. Where the calculi are small, copious hot enemata may cause their expulsion. Violent purgatives should be avoided if calculus is suspected, since they would only intensify the pain, and may lead to rupture of the bowel. Surgical operations for calculus have been recommended, but are not justified by results.

Dung-balls.—These are collections of ingesta which may be met with in some part of the colon, large or small, or in the rectum. They are globular in shape, firmer than normal ingesta, and are usually larger than the average calculus. They give rise to obstruction of the intestines, and may lead to impaction or less frequently to enteritis.

Symptoms set up are those of subacute colic with impaction, and they are usually fatal on account of their large size and the impossibility of removing or reducing them. Diagnosis is assisted by exploration per rectum.

Treatment is generally of no avail unless the dung-ball is rectal in position. Most of the animals affected succumb to the resulting impaction. The measures to be adopted are the same as for impaction of the colon, consisting of the administration of intestinal stimulants and antispasmodics, to be followed by a purgative. If the obstruction is in the rectum, it may be broken down and removed piecemeal, with the assistance of copious hot enemata.

Impaction of the Intestines in the Horse.—This condition consists of an accumulation of hard, dry, partly digested material in some part of the intestinal tract. It may also be brought about by sand, and occasionally in the small intestines by the presence of enormous numbers of parasites. The large intestines are by far the most frequently affected, particularly in the region of the double colon, but impaction may also occur in the small intestines and also in the cæcum and small colon.

Causes of the Condition.—This condition is most often brought about as the result of overfeeding on improper foods, especially those containing large proportions of indigestible fibre, such as oat chaff, chopped straw, hard dry clovers, and the old dry grass in a burnt pasture towards the end of a hot summer. It is predisposed to by lack of sufficient exercise and insufficient water supply. Sand may also be responsible for the impaction, particularly when horses have been fed for a considerable time on foods containing sand and also drinking from shallow ponds with a sandy bottom. It gives rise to a condition which has been described as *sand colic*. The common situation of the sand is the large intestine, principally the cæcum.

Impaction may also be predisposed to by certain intrinsic causes, such as defective muscular power of the intestinal wall (atony or paralysis), defective secretions, stricture, tumours pressing on the bowel, calculi, and dung-balls. It is more frequently met with in old animals than young, and this is probably due to defective peristalsis, and to irregular teeth interfering with mastication and impairing digestion.

Impaction of the Small Intestines.—This condition is exceedingly rare, but it may be brought about by greedy feeding in horses that have been kept for some time without food. In other cases it may be due to sand. Impaction has also occurred from a huge accumulation of round worms (*Ascaris megalocephala*). The obstruction is sometimes met with in the duodenum, more rarely in the jejunum, and rather more frequently in the ileum close to its termination at the cæcum.

Symptoms.—These are variable according to the position of the impaction. When the duodenum is affected, the symptoms develop rather rapidly within a comparatively short time after feeding, giving rise to acute colic, with severe abdominal pain. When the ileum is affected, the symptoms do not develop so rapidly, and it often happens that there is an interval of several hours since the last feed. The pulse is quick and soft and lacking tone, the mucous membranes are usually pallid, but occasionally injected or even dirty-looking. A small amount of fæces may be passed from time to time for several hours; after this there is a cessation in the passage of fæces. The pain is indicated by frequently looking round at the sides, and kicking forward at the belly. The patient lies down and rolls and gets up again, pawing the ground. As the condition advances, the pain may get more acute and the pulse becomes more frequent, smaller, and sometimes difficult to feel. Gastric tympany may also be set up. The condition may be distinguished from impaction of the large intestines by examination per rectum, but apart from that definite diagnosis is very difficult, unless the impacted portion of the small bowel is actually felt.

Treatment.—If impaction of the small intestines is diagnosed, a purgative may first be given, such as aloes, or a combination of aloes and calomel. This should be followed by the administration of alimentary and general stimulants, such as carb. of ammonia, nux vomica, and turpentine. The latter may be given in milk if a purgative has been previously administered, if not it may be administered in oil. If the pain is very acute, and is not relieved by these measures, chloral hydrate may be administered. It is best not to give opium or its derivatives, inasmuch as they reduce both secretions and peristalsis, and thereby tend to intensify the condition. Relief of pain may also be afforded by the injection of copious hot enemata, and by the application of hot cloths or counter-irritants to the abdominal wall. If relief is not obtained within a few hours, lavage by means of the stomach tube should be resorted to (see p. 305).

Impaction of the Colon.—The large colon is the most common seat of impaction in the horse. Impaction of the colon gives rise to a form of subacute colic which may last for several days, terminating either in relief or death. The impaction is generally due to ingesta, occasionally to sand, and is predisposed to by improper and irregular feeding with bulky foods containing large proportions of indigestible fibre. Defective secretions and defective muscular power may also be responsible for the condition, as may also a definite obstruction, such as calculus or dung-ball. It is comparatively rare to meet with several cases of impaction simultaneously in a stud of horses that are fed on similar lines. It is therefore probable that the intrinsic causes are of greater importance than the nature of the food. The stagnation of ingesta in this viscus causes it to become dry and firm, owing to the absorption of liquids from it. It only occasionally results in fermentation with tympany.

Symptoms.—Symptoms of impaction of the large colon are those of slowly developing subacute colic. It commences with dulness and depression, with capricious appetite and some abdominal discomfort. The pulse is soft, large, and sluggish. The temperature is not interfered with. During the first twenty-four hours or so fæces are passed in smaller quantities than normal, and are often coated with mucus. Defæcation then ceases. Abdominal pain becomes more pronounced, the horse frequently looking round at his flanks, pawing with his feet, and kicking forward of the abdomen. As the horse is still feeding intermittently the intestines tend to become more and more distended. The pain is then increased and more violent, and the pressure of the impacted colon on the diaphragm causes some slight inspiratory dyspnœa. Pressure of the colon on the bladder causes frequent attempts at micturition. The abdomen is somewhat distended, but is not in any way resonant as in flatulent colic. Examination per rectum is of the greatest importance in diagnosing this condition. The anus is generally loose and flexed, and the rectum more or less "ballooned." The impacted colon can also be felt in the form of a firm mass into which one can make digital impressions. The colon may be almost wedged into the brim of the pelvis, particularly when the impaction is in the pelvic flexure of the colon. Auscultation of the abdominal wall reveals the absence of peristalsis. In some cases the symptoms suddenly become very acute, the animal breaking out into patchy sweats, lying down, rolling and getting up again in quick succession. When the condition has been in existence for several days some tympany may occur. When the impaction is due to feeding on new oat chaff, as is the case sometimes in agricultural horses in threshing time, the impaction is generally accompanied by tympanites and acute colic, and is often followed by enteritis and diarrhœa.

Treatment.—It is well first to begin with a full dose of aloes administered in the form of a ball, or, instead, repeated doses of linseed oil Oii. may be given with ol. tereb. ℥ii. and ether sulph. ℥i. The hypodermic injection of liq. strychnia ʒi.-ʒii. is of very great assistance in stimulating peristalsis. The pain may be controlled by chloral hydrate ℥i., administered in water Oi. If there is no action of the bowels during the following twenty-four hours, the hypodermic injection of eserine and pilocarpine āā gr. i. should be given, or the pilocarpine might be administered ten or fifteen minutes before the eserine, since it mainly stimulates secretions; eserine being given afterwards causes less pain from the exaggerated peristalsis it sets up. If there has not been a previous attempt to reduce the consistence of the ingesta there is the danger that eserine might cause rupture of the bowel.

The best results, however, appear to be obtained by the treatment suggested by Caulton Reeks. He first administers the following in the case of a cart-horse:

Ammon. carb. ℥ii.
Nux vomica ℥i.
Sapo. mollis q.s.

This is made into four balls to be administered at once, and is followed by a draught consisting of ol. tereb. ℥ii., spts. ammon. arom. ℥ii., ol. lini ad Oi. If the animal remains in pain, this treatment is repeated in from three to six hours later, omitting the nux vomica and substituting pulv. zingib. If this does not afford relief, and if the pulse is strong, he next injects hypodermically eserine sulphate gr. i., and if there is still no action of the bowels after a reasonable interval he repeats the eserine in a larger dose of gr. ii. In the case of light horses and young cart-horses, he reduces the dose of nux vomica to ℥ss. This line of treatment at first would appear to be somewhat heroic, but Reeks's experience, together with that of the writer and many others, has proved its efficacy. Ill-effects from the large doses are never observed. Enemata must also be administered, and they should first be given in medium quantities, frequently repeated, at about blood heat, in order that they may be retained and assist in softening the ingesta. Several hours later the enemata should be hot and copious, and injected as far as possible by means of a Read's pump. These both relieve pain and stimulate peristalsis, and assist in the removal of the ingesta softened by the previous enemata. Where there is tympany it must be relieved by the use of the trocar and cannula. The application of hot cloths covered with a mackintosh sheet, or the application of mustard, assists very materially in reducing the pain. After-treatment consists in attention to the diet, which must be soft and mucilaginous for several days, the normal diet only being returned to after peristalsis is well established and the animal very much brighter. It should be accompanied by a course of vegetable tonics, such as gentian, nux vomica, and calumba.

Impaction of the Cæcum.—This is not a very common condition except as a complication of impaction of the colon. It may, however, occur without that portion of the bowel being involved. The cæcum is also sometimes impacted with sandy material taken in with drinking-water.

Symptoms are those of subacute abdominal pain, the animal lying down for long or short periods. The depression lasts for several days, and may prove fatal in from seven to fourteen days. The appetite is fair throughout the earlier part of the attack, and the fæces are passed frequently and in small amounts. In some cases in the earlier stages diarrhœa actually exists. The condition is very difficult to diagnose definitely, and suspicion is as a rule only confirmed on post-mortem examination.

Treatment.—As the diagnosis in a majority of cases is that of subacute colic, treatment is accordingly based upon the relief of pain. The administration of chloral hydrate or of turpentine in oil may be resorted to. If there is good reason for suspecting the actual condition, then stimulant treatment should be adopted as for impaction of the colon in the horse. The majority of cases, however, are fatal.

Impaction of the Small Colon.—This condition is brought about by the same causes as impaction of the large colon, namely, defective secretions and defective muscular power associated with improper foods.

Symptoms commence with subacute colic with pain more or less continuous, which gets more severe as time passes. Attempts at defæcation are after the first few days in vain, and there may be some eversion of the rectum. Examination per rectum may reveal the impacted bowel, but in other cases it is forward in the abdomen and cannot be located. The condition is often fatal in from two to four days. A definite diagnosis is very difficult, unless the impaction can actually be felt per rectum.

Treatment should be based on the same principles as impaction of the large colon. A dose of aloes followed by the stimulant line of treatment is most likely to be successful along with frequently repeated copious enemata. The condition is, however, fatal in the majority of cases.

Twist of the Bowels ; Volvulus ; Incarceration ; Strangulation.—These conditions probably constitute the largest proportion of fatal cases of colic in the horse. They vary somewhat anatomically, but the conditions they produce correspond very closely pathologically. Incarceration or strangulation consists of the arrest and constriction of a piece of bowel through a natural or accidental orifice, which results in interference with the circulation through the part, as in the case of a strangulated hernia. Volvulus or twist may involve either the bowel, which may become twisted upon itself, or the mesentery, which may become screwed round and so impede the circulation to the bowel. In either instance the interference with the circulation in the affected bowel permits the invasion of organisms into the bowel-wall and through it into the peritoneum. Gangrene may also be set up, and thus toxins are produced which become absorbed with fatal results. Death is frequently thought to be due to pain and shock, but although these may be contributory causes it is more than probable that the absorption of toxins is the main cause of death.

Twist may occur in the small intestines or the large intestines. When the large intestines are affected the condition is usually only a *partial twist*, consisting of a displacement of the bowel, producing a kink which partly occludes the blood-vessels, and also causes some arrest of the ingesta, thus leading to partial impaction. This most commonly affects the large colon, particularly at the pelvic flexure. It may also occur at the diaphragmatic flexure. A similar condition may occur with the cæcum, the point of which may become displaced or partially folded, with interference with the circulation through the viscus. The severity of the condition will depend upon the acuteness of the kink and on the degree of obstruction of the vessel. It is possible in some instances that the displacement may ultimately correct itself and recovery occur, but the majority of these cases are fatal. The small intestines are more commonly affected with volvulus, which in this instance is usually a *complete twist.* The common starting-point in these cases is a small rent or rupture of the mesentery, through which a loop of bowel passes. This acts as a pivot or turning-point, and by exaggerated peristaltic action the bowel becomes twisted round and round a number of times, in this way occluding the mesenteric vessels and causing a complete strangulation. In other cases no rupture of the mesentery is discoverable, and the starting-point of the twist would appear to be a loop of bowel which may have become distended with gas. The extent of these twists of the small bowels is often very extraordinary; the knot resulting may be so complex as to almost defy unravelling even when the bowel is spread out on the floor during a post-mortem examination. Needless to say these cases are invariably fatal. The commonest portion of the small intestine involved is the jejenum, while twist not infrequently occurs in the ileum near to the cæcum, and only extremely rarely in the duodenum.

Causes of Twist.—The causes of twist are very obscure, and the subject has led to much speculation. It may be brought about by some unexpected sudden movement such as a fall or a false step, the slip bringing about a sudden dislodgement of ingesta in the large intestines and causing a displacement. A similar fall may also cause a rupture of the mesentery, which, as already pointed out, may be the starting-point of the torsion. That this is undoubtedly the case is clearly indicated by the clinical history of some cases. It has happened that a horse, apparently perfectly well, in turning round a corner has slipped and fallen. He has risen again and gone on with his journey, and within half an hour has begun to show signs of acute colic. The symptoms have developed rapidly, and death occurred within twenty-four hours, post-mortem examination revealing the presence of a twist of the small intestine with a small rupture of the mesentery. The matter is of considerable importance from a legal standpoint, since the death in such a case is undoubtedly due to the accidental fall, and if the horse happens to be insured there is a good case made out for claim under the "accidental" policy. Twist may also be due to acute irregular peristalsis, such as may be brought about by irregular feeding. One commonly meets with cases in horses that have been for an unusually long journey and have not been fed during that time, and have been brought back to the stable in a more or less exhausted condition. They have then been fed in the ordinary way, and some time later acute colic sets in, which ultimately proves to be twist. It is possible, too, that the presence of worms (ascarides) may also set up irregular peristalsis resulting in twist. This is suggested by the frequency with which such worms are found in the intestines of horses dead of twist. One must acknowledge, however, that such worms are extremely common in horses, and are found with great frequency in autopsies on animals dead from other conditions. The presence of worms in twist may therefore only be coincidence. Rolling during attacks of colic arising from other causes is often thought to be responsible for twist, particularly partial twist. This may be so, but it is probably not a common cause of twist. One frequently sees horses rolling vigorously when turned out to grass, but it is almost unknown for twist to follow. It is much more probable that the presence of twist is responsible for the rolling in fatal cases of colic rather than the rolling is responsible for the twist.

Symptoms of Partial Twist.—This is the form which usually involves the large intestine—colon and cæcum. The symptoms are usually ushered in by subacute abdominal pain, which is continuous and may last for several hours. Now and then there may be more acute spasms, which pass off again, the dull pain continuing. The mucous membranes are injected, the pulse and respirations accelerated. There is a passage of fæces in small quantities, often accompanied with flatus. The temperature may be elevated up to 103° F. or 104° F. The horse frequently lies down and may roll and rise again, but there is rarely much violence. As the condition advances the animal breaks out into perspiration, and frequently becomes tympanitic. Examination per rectum may afford considerable assistance in diagnosis. The anus is usually tightly constricted and the patient objects to the exploration. In passing the arm into the rectum the bowel is found to cling more or less to the arm. The pelvic flexure of the colon as a rule cannot be felt owing to the displacement. Recovery from this condition may occur, the displacement being reduced spontaneously, either by rolling or by the contractions of the bowel-wall. In fatal cases the animal becomes weaker and staggers, and may ultimately fall and die from exhaustion and the absorption of toxins in from one to three or four days.

Treatment of Partial Twist.—This consists of the same principles as for subacute spasmodic colic. The patient should be stimulated by means of ammonia and ether to which ol. tereb. may be added in cases accompanied by flatulence. Hot cloths should be applied to the abdominal wall, and copious hot enemata injected. These, however, are almost immediately expelled, but perhaps not quite so rapidly as in complete twist. Tympany should be relieved by the use of the trocar and cannula. There is no objection to allowing the horse to roll, provided he is not violent and does not injure himself. On the other hand, it is possible that moderate rolling may assist in reducing the partial twist. The administration of violent purgatives such as eserine is recommended by some authorities, but there is much to be said against it in that in any case of twist such drastic remedies serve only to intensify the pain.

Symptoms of Complete Twist.—This form is usually met with in the small intestines or the floating colon. The condition is very serious, causing very acute, more or less continuous spasmodic colic, and is invariably fatal. The horse knocks himself about considerably, frequently lies down and rolls, may remain on his back with all his legs in the air, a position which sometimes appears to afford temporary relief. The pulse at the first is usually full and hard, but soon becomes small and wiry. Respiration is blowing and accelerated. The temperature at first is usually normal and gradually rises owing to the excitement and pain. The horse breaks out into profuse patchy perspiration. When standing he often does so with his hocks and fetlocks flexed and his fore-limbs widely apart. In some instances there is marked flatulence, but tympany is not by any means a constant symptom. When it does occur the use of the trocar and cannula sometimes affords very great assistance in diagnosis, as the withdrawal of the trocar may be followed by a jet of blood. This would appear to be one of the few almost diagnostic symptoms of complete twist, but is unfortunately only available in a small proportion of cases. Examination per rectum may assist in diagnosis, the anus usually being tightly constricted and the rectum empty and contracted. The cord-like twisted mesentery may sometimes be felt. Enemata are expelled immediately. Death from complete twist may occur in from ten to thirty-six hours.

Treatment.—There is no known method by which complete twist can be reduced. Surgical interference, even in the case of positive diagnosis, would be useless, as might have been gathered from the previous statement that it is almost impossible to unravel a complete twist even when one can spread out the intestines. Positive diagnosis, however, is almost impossible, and treatment, therefore, is based on the same lines as for acute spasmodic colic, the greatest reliance being placed upon powerful sedatives and antispasmodics such as chloral hydrate and ether, together with the application of hot cloths or counter-irritants to the abdominal wall and the administration of frequently repeated enemata. If the patient is very violent it may be impossible to administer medicines *per os*, and in such cases morphia in large doses, gr. vi.-viii., may be given. It seems, however, almost impossible to afford much relief by any means when complete twist is actually in existence. If a definite diagnosis of complete twist could be made, by far the better plan would be to slaughter the patient at once, but owing to this impossibility, and the fact that other forms of acute spasmodic colic may give rise to similar violent symptoms followed by recovery, such a course is obviously impossible. Preventive measures should be adopted in studs where "outbreaks" of twist have occurred. In such instances it is certain that the condition has been caused through bad horse management, with particular reference to feeding and watering. Consequently attention must be drawn to the necessity for frequent and regular feeding with sound foods and to the watering of horses more frequently, even on prolonged journeys. (See paragraph on "Causes of Colic.")

Intussusception ; Invagination ; Telescoping.—This is a condition in which one portion of the bowel becomes telescoped into another portion of the bowel. It is usually the anterior portion passing into the part behind it. Occasionally, but rarely, intussusception is in the reverse direction. In the horse the most common seat of it is the jejunum, and it results in serious interference in the circulation of the part, with congestion and inflammation particularly of the peritoneum. It may also result in necrosis and gangrene of a portion of the bowel. Occasionally the affected portion is the ileum, which may pass through the ileocæcal valve into the cæcum or the colon. The cæcum also rarely becomes invaginated into the colon. Still more rarely the small colon is involved, either being invaginated into the rectum or forward into the large colon.

Causes.—This condition is probably due to irregular peristalsis, which may be brought about from various causes, such as inertia of a portion of the bowel, simultaneously with spasm of an anteriorly adjoining portion (Cadeac). Sometimes the irregularity is due to worms, and at other times to sudden exaggerated and unexpected movements, as in falling.

Symptoms.—These commence as a rule with subacute abdominal pain, which in the horse, however, rapidly becomes acute and assumes the character of spasmodic colic (see *ante*). In few instances there may be blood passed with the fæces. There is rarely much tympany, and death may occur in eight to twelve hours after the onset of symptoms. When the cæcum is invaginated the symptoms are much more prolonged, and may last for several days.

Treatment.—This is not very hopeful, but owing to the difficulty of definite diagnosis the condition would be treated as acute spasmodic colic, relying principally upon anodynes and antispasmodics internally and counter-irritants externally.

Verminous Colic ; Colic due to Verminous Aneurism.—This form of colic is met with more particularly in young horses. It is due to aneurism and thrombosis of the anterior mesenteric artery, brought about by the immature stage of the *Strongylus vulgaris* (*Sclerostomum vulgare*). The parasites, according to Sticker, attain this position by way of the general circulation, minute larvæ becoming arrested in the vasa vasorum. Here they increase in size, and produce a lesion of the intima of the vessel wall resulting in the formation of a thrombus. The increase in size of the thrombus, together with the weakening of the arterial wall, leads to dilatation and the development of an aneurism, and may cause complete obstruction to the circulation through the vessel. If the vessel involved is the main artery, fatal results are produced, but if it occurs in one or more of the branches of the artery, it is less likely to prove fatal, owing to the collateral circulation.

Symptoms.—When the anterior mesenteric artery is involved, there is a sudden appearance of acute abdominal pain, which increases in violence, and may last for several hours. The animal knocks himself about violently, rolls and struggles. There are frequent attempts at defæcation and urination. The pulse is quick and hard, and the mucous membranes injected. Respiration is blowing, and the animal breaks out in patchy sweats. Death may occur in from four to twelve hours in convulsions. In those cases where the smaller vessels are involved the symptoms come on more gradually and are not so violent. Acute colic, however, may last for several hours and then develop into continuous subacute abdominal pain. This form is less likely to be fatal, but may continue to produce symptoms for days or weeks.

Prognosis is exceedingly difficult in either case, but the rapidity of the course in the acute form does away with the necessity for prognosis. If death does not occur in a few hours and subacute colic supervenes, it is quite impossible even then to foretell the termination of the affection. In cases where recovery takes place intestinal lesions are produced by the parasites passing down the mesentery to the bowel wall. This results in debility and emaciation, and may terminate either in death or recovery.

Death in verminous colic may be due in the acute stages to direct interference with the circulation of the bowels, with the onset of gangrene and the absorption of toxins. In the subacute cases death generally results from the enteritis which follows the interference with the circulation, and the passage of the parasites into the intestinal wall.

Treatment.—There are no curative measures that can be applied to the relief of this condition, and one must devote one's efforts to reducing the symptoms of pain. The best agents are morphia hypodermically or chloral hydrate or cannabis indica, which may be drenched by the mouth or given by the rectum as an enema.

G. H. W.

THE TREATMENT OF GASTRO-INTESTINAL DISORDERS IN HORSES BY LAVAGE

Use of the Stomach Tube.—Lavage by means of a tube introduced into the stomach, by either the nasal or oral route, offers an additional aid in the treatment of gastro-intestinal affections associated with fermentation and impaction. In addition, the tube is of great service in the treatment of the condition known as choking, especially that form of choking produced by chaff and crushed grain, where the food material has lodged in the thoracic portion of the œsophagus. Stomach-tubes may be passed either by the mouth, in which case it is usual to employ a double tube with inlet and outlet tubes, or by the inferior nasal meatus, where a single tube of smaller diameter is more suitable. Both types of tubes have their advocates, their advantages and disadvantages, and it is largely a matter of individual opinion as to which should have the preference. The advantages of the double tube are the possibility of using a tube of greater calibre and the slightly quicker emptying of an overloaded stomach. Against these are the greater initial cost, the greater liability to become injured by the teeth, the greater resistance by the patient to its passage, and the shorter life of the tube. The single tube, on the other hand, costs less to begin with, lasts longer, is exposed to no risk of injury, is not resented by the patient, and will do all that a double tube will do, but the accomplishment may take slightly longer. The passing of a suitable single tube by way of the nostril and pharynx is quite an easy and safe procedure when once the technique has been mastered.

This brings us to the consideration of the tube itself. The quality of the tube is most important. It should be glass moulded and of pure rubber without canvas stiffening. It must be fairly stiff and yet supple enough to follow the contours of the nasal meatus and to do no injury to the soft structures. Its length is about nine to ten feet and its diameter about five-eighths of an inch. At one end the tube is smoothly tapered and rounded off, at the other extremity a metal connection is fitted for attachment to an enema pump. About eighteen inches from the rounded end the tube is marked, and again at about five and a half feet is another mark. The first mark gives an approximate guide as to the distance to the pharynx; the second informs the operator that the tube is approaching the stomach.

The double tube, which on account of its greater calibre can only be passed by the mouth, consists of a rubber tube about one and a quarter inches in diameter with a smaller tube moulded into its wall, except for a length of about eighteen inches, where it is free, and to the extremity of which the pump is attached. The large tube is the outlet tube and the small one the inlet. The technique of passing the double tube is simple; the jaws are held open by means of a speculum and the tube passed into the œsophagus in the same manner as an ordinary probang, or in place of keeping the jaws open they may be strapped together and the tube inserted through the interdental space. After the tube has entered the pharynx the same technique as is used for passage *via* the nasal route is carried out, as will be described later.

Before attempting to describe the technique of passing the tube *via* the nares it is necessary to consider the anatomy of the nasal chambers and the physiology of the act of deglutition, because it is upon a working knowledge of these that the passing of the tube to the stomach is made possible, safe, and easy. The nasal chambers are divided into three meatuses or channels by the turbinated bones, the anterior openings of the middle and inferior or floor of the chambers are accessible from the nostrils. It is here that the first error in attempting to pass the tube is likely to be made. If the middle meatus be traced backwards it is found to terminate against the ethmoid bone, and there is no wide foramen leading to the pharynx, consequently the tube, if introduced into the middle meatus, is stopped at the ethmoid and can go no farther. The inferior meatus, however, terminates in a wide foramen opening directly into the pharynx, and consequently the tube, when pushed along the floor of the nasal chamber, glides easily and without resistance into the pharynx. The superior meatus may be disregarded as being both inaccessible from the nostril and on account of its small diameter, so that it is next to impossible to introduce the tube into it.

Swallowing is a combination of voluntary and involuntary action, the first being the working of the food over the base of the tongue and into the pharynx, where voluntary control ceases and the remainder of the act becomes involuntary. Briefly, the nervous mechanism of deglutition is as follows: the afferent impulses set in motion by the presence of food, or, in our case, by the presence of the tube, proceed *via* the fifth and superior laryngeal nerves to the swallowing centre in the medulla; from this centre efferent impulses are sent to the pharyngeal muscles, larynx, and œsophagus, by way of the pharyngeal plexus, glossopharyngeal, hypoglossal, and recurrent laryngeal nerves, the net result of this reflex action being the closure of the glottis, constriction of the larynx, and, the most important from our point of view, the advance of the pharynx and œsophageal infundibulum which seizes the food or tube, as the case may be. Waves of peristalsis now run down the œsophagus, carrying the food or tube into the stomach.

Technique: Position of the Patient.—This is of some importance to the easy passage of the tube. The head should be well extended and in a straight line with the neck. The head should be steadied by the assistant or attendant placing one hand on the neck and the other on the nose well above the nostrils. The assistant should stand on the opposite side to that of the operator and should maintain his hold of the patient throughout the entire operation and endeavour to prevent any sudden movements. The use of the twitch is both unnecessary and undesirable and should be avoided wherever possible. As a rule, the patient offers no resistance to the manipulations, which, if carried out properly, cause very little, if any, inconvenience and no pain. In very exceptional cases one meets with violent or vicious animals that will completely defeat all attempts to handle the head until brought under the influence of a narcotic, but as a rule the animal is too ill to make any resistance, even if vicious.

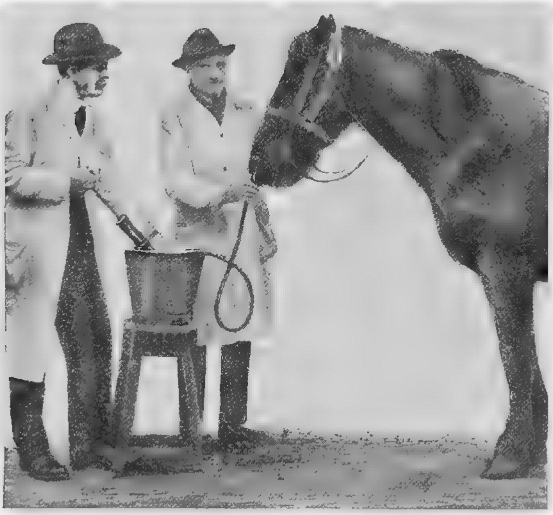

FIG. 74.—Stomach-tube passed by nasal route, the horse standing perfectly quiet and offering no resistance.

Position of the Operator.—The tube can be equally well passed by either nostril and the choice lies with the operator, it depends upon his ability to use either hand with equal facility. The left nostril is, however, usually selected, but if the right is chosen all positions as described are simply reversed. The operator stands on the near side of the patient, slightly in advance of the nostril, the rounded end of the tube in the left hand, and the remainder of the tube hung around the shoulders. In cold weather it is advisable to place the tube in a pail of warm water before use to prevent cracking and checking and also remove any undue stiffness due to the cold. The first eighteen inches or so should be lubricated with linseed tea or decoction of slippery elm bark or, in cases of emergency, with the patient's own saliva, and the remainder of the tube should be lubricated as it is passed in. Vaseline, grease, or oils should not be used owing to their injurious action upon the india-rubber of the tube. The left hand now introduces the end of the tube into the left nostril, the fingers of the right hand depressing it and guiding it into the inferior meatus or floor of the nasal chamber. The left hand then gently pushes the tube in until the first mark appears or a swallow occurs, which indicates that the tube has passed into the pharynx. The tube should now be withdrawn a couple of inches and the fingers of the right hand applied to the exterior of the pharynx, the left hand grasps the tube about four inches from the nostril and cautiously advances it until the right hand detects the commencement of a swallow, when the tube is rapidly pushed forward to meet the advancing œsophageal infundibulum, which, if the manipulation has been successfully carried out, grasps the tube, giving the sensation of a distinct tug. The tube is then gradually fed in as swallow succeeds swallow until the stomach is reached. The successful passing of the tube depends entirely on the act of deglutition; in fact, once the tube is grasped by the œsophagus it is in reality swallowed and all the operator has to do is to feed it in, no force at any stage of the procedure is required, and is only productive of harm and possible injury. Usually as the tube approaches the stomach (as indicated by the mark on the tube) some slight resistance is felt; this is overcome by steady but gentle pressure and the entrance to the stomach is effected.

Accidents and Difficulties.—The accidents that may occur during attempts to pass the tube or when withdrawing it are in nearly all cases quite avoidable if it be borne in mind that no force is required, and the proper method of withdrawing the tube adopted. If the tube is entered into the middle meatus by mistake and pushed in rapidly the ethmoid bone is liable to injury or fracture and a profuse hæmorrhage results. Fracture accompanied by more or less severe hæmorrhage of the inferior turbinated bone may also occur if the tube is pushed in too rapidly after it has entered the œsophagus, from the tube kinking up and impinging on the bone. It can also happen if when withdrawing the tube the end flies up and strikes it. Both these accidents can be avoided by first of all keeping time with the swallows, and secondly, when withdrawing the tube by keeping it well down on the nasal floor with the fingers of the right hand while pulling it outwards and upwards with the left hand. The patient nearly always gives his head a jerk as the tube leaves the nostril, and unless the tube is steadied as described the end is bound to fly up. When hæmorrhage does occur from either of these accidents it is quite profuse and somewhat alarming to the client, but usually yields readily to cold douching or the general line of treatment for epistaxis.

A difficulty that sometimes occurs, generally from a tube that is too supple, is the passing of the tube into the larynx instead of into the œsophagus; this, as before stated, may be the fault of the tube or by the operator " missing the swallow," or, in other words, either pushing the tube too far before the advancement of the infundibulum, or too late for it to be caught by it. In old tubes a stilette may with advantage be used in the tube in order to stiffen it, but the stilette should be withdrawn as soon as the tube enters the œsophagus. To detect whether the tube has entered the larynx or not, the free end should be applied to the ear, when, if such an accident has occurred, the expired air will be felt against the cheek. The occurrence of coughing when passing the tube is no reliable indication as to its whereabouts, as contrary to expectation the entrance of the tube into the larynx does not invariably excite coughing, whereas the passage of the tube into the pharynx very often does. It will thus be seen that the expired air is the chief indication that the tube has gone the wrong way. Again, when the tube passes into the larynx, no tug or resistance is felt as in the case when it enters the œsophagus.

Clinical Applications and Indications for the Use of the Stomach-tube

Diagnosis.—For diagnostic purposes in gastro-intestinal disorders the tube is of considerable value, as the presence of gas or otherwise in the stomach and the character of the gastric contents are easily and definitely ascertained by the passing of the tube and siphonage of the stomach. It is also possible during the course of intestinal disorders to discover whether anti-peristalsis with the entrance of ingesta into the stomach *via* the pylorus is taking place. This, in the writer's experience, is a very common occurrence in protracted cases of flatulent colic, and is responsible for recurrence of the intestinal tympany and the production of the gastric symptoms so often associated with the later stages of flatulent colic.

Acute Gastric Tympany.—This condition is the one *par excellence* for the immediate use of the tube. It may be necessary, it is true, first of all to bring the patient well under the influence of a hypnotic or narcotic, but once under control the tube should be passed without delay. Upon entering the stomach there is a gush of gas and liquid chyme from the free end. The patient evinces immediate relief and the effect upon the heart's action is very noticeable. In order to prevent the fresh generation of gas in the stomach, lavage should now be practised. The free end of the tube is attached to an enema pump, and from one to two gallons of warm water in which has been placed an ounce of creolin or some other harmless antiseptic are pumped slowly into the stomach, the tube is disconnected from the pump and the free end allowed to hang down towards the floor, thus siphoning off the injected fluid. Should the flow cease before all that has been pumped in has returned, the pump is reattached and some more pumped in, as the tube has probably become blocked by ingesta. This alternate filling up and siphonage of the stomach should be repeated until little or no fluid returns, which indicates that the stomach is not only empty but that the " pyloric lock " has become relaxed, allowing the fluid to proceed into the intestines. Any medicines that it is thought desirable to exhibit may now be given by way of the tube, or about two gallons of warm water containing an antizymotic may be pumped in. It has been found that lavage, combined with such medicinal treatment as the case may indicate, solves the hitherto difficult and unsatisfactory treatment of acute gastric tympany.

Impaction of the Stomach.—In this serious condition lavage again offers the most satisfactory means of treatment, but it is a far more laborious and lengthy procedure than in tympany. Repeated and continued injections and siphonings are necessary before the impacted mass is sufficiently softened and broken up to allow of its passage into the intestine or evacuation through the tube, but the results are well worth the extra time and effort and, even if it is not possible to empty the stomach completely at one sitting, great relief is obtained, and the injection of an antizymotic will keep down fermentation until the next day, when the attempt to empty the stomach may be resumed.

Acute Intestinal Tympany or Flatulent Colic.—Lavage again finds a useful place in the treatment of this condition, and is carried out in the same manner as above described with the exception that it is advisable to pump in from four to five gallons of warm medicated water after the preliminary stomach lavage. Lavage combined with nerve stimulants very quickly induces resumption of peristalsis, when the exhibition of a quick-acting purgative such as eserine or arecoline and copious enemata soon promote evacuation of the bowel contents and a subsidence of the symptoms. In cases that are seen early and before the distension of the large bowels has become excessive, lavage obviates the necessity of puncture by the trocar, the tympany rapidly subsiding and very seldom recurring. Unless very free purgation is thought advisable, no more than five gallons of water should be introduced into the system; this of course does not include the water used in the actual lavage, which may amount to many gallons before the stomach is emptied.

Subacute Obstructions and Impactions of the Large Intestines.—Lavage has a special place in the treatment of these conditions, as it not only ensures a clean stomach but is the means whereby large quantities of fluid can be introduced into the bowels with the object both of softening the impacted mass and of stimulating and helping to restore the lost tone of the intestinal walls. It overcomes one of the most pronounced and unfortunate symptoms of these conditions, viz. the obstinate refusal on the patient's part to drink. By means of the injection of large quantities of medicated water, fermentation is kept at a minimum and elimination by the kidneys stimulated, thereby decreasing the auto-intoxication which is one of the complications of the condition. The stomach is washed out in the usual way two or three times daily, and at each operation four to five, or even more, gallons of warm medicated water are pumped in and allowed to remain. To help to soften the impacted mass, hyposulphite of soda may be administered in divided doses —twelve ounces being given daily. Nerve stimulants and copious rectal injections are useful to stimulate peristalsis, which when established again may be further increased by subcutaneous injections of arecoline. In cases that are amenable to any treatment the course of the affection is materially shortened, three or four days being sufficient to establish normal functions.

Spasmodic Colic.—At first sight it may not be apparent how the use of the stomach-tube can be of any service in this condition, especially as many colics of this type recover without any treatment whatever; but when it is considered that indigestion is probably the most frequent cause of spasmodic colic and that stasis of the intestinal contents is responsible for the occurrence of the spasms, lavage of the stomach and the injection of an antizymotic and antispasmodic seem to be most logical. Such indeed is the case, and spasmodic colic yields most satisfactorily and rapidly to the washing out of the stomach and the exhibition of a gallon of warm water in which an ounce of chloral hydrate has been dissolved.

Choking or "Choke."—In the condition known as choking the use of the stomach-tube plays an important part, being much more effectual and safer than the probang. There are two methods in which it can be employed. The first, a very crude but often very effectual method, but only applicable under certain circumstances, consists of passing the tube down to the obstruction, depressing the head nearly to the floor and forcibly pumping water against the obstruction. This in the cases of chaff or crushed grain, bran, etc. will often loosen up the mass sufficiently to allow it to pass onward towards the stomach; some of it will of course be evacuated through the tube. Care must be taken to keep the head low or there is a danger of the water or food materials passing into the larynx and trachea. The second method consists in passing the tube down to the obstruction and very cautiously pumping in a small quantity of water. In mild cases the softening action of the water and also its weight will be sufficient to cause the mass to pass on into the stomach. If, however, success is not obtained after a few attempts and the symptoms are urgent the œsophagus and contained tube should be pressed outwards in the jugular furrow and an incision about two inches long made so as to expose the œsophagus but not to wound it. The incision can be made under local anæsthesia, is made longitudinally to the œsophagus, and, needless to say, with all surgical precautions as to asepsis. A wide piece of tape is now to be passed round the œsophagus above the obstruction and tied firmly but without injury to the soft structures. Water is now pumped into the œsophagus until it is filled up to the ligature, the pump is disconnected and the fluid allowed to siphon off. This is repeated until the mass either passes on or is removed by the continued siphoning. In many cases the weight of the column of water and the dilatation of the œsophagus are sufficient to cause the obstruction to move on; otherwise, unless of course the offending object is a solid body such as an apple, potato, or egg, etc., siphonage is necessary to complete its removal. The ligature is now untied and the tube withdrawn, the wound is treated antiseptically and is best left unsutured. There appears to be no real danger of injuring or rupturing the œsophagus during the above manipulations, but care should be taken not to distend it too forcibly when pumping the water in, as it is conceivable that if the walls were weakened by chronic distensions or ulcerations they might give way, and the possibility of such an accident happening should not be lost sight of.

C. G. S.

Colic in Cattle

Abdominal pain in cattle is frequently met with, but the symptoms set up are never so acute and rarely so serious as in the horse. Most of the causes which give rise to symptoms of abdominal pain in ruminants are stomach affections, such as impaction, tympany, and foreign body in the reticulum, but these conditions are not usually included in the term colic. The commonest causes of colic are uterine affections in the female, obstinate constipation, intussusception, volvulus or twist, "gut-tie," and also nephritis and the presence of calculi in the urinary tract. These conditions all set up a form of spasmodic colic which may be either acute or subacute.

Spasmodic Colic in Cattle.—This may be set up by any of the conditions already enumerated.

Symptoms.—The symptoms of spasmodic colic in cattle consist of general uneasiness, paddling of the hind feet, stamping, kicking of the abdomen, looking round at the sides and lashing of the tail. There is frequently a staring expression in the eyes, and the animal may become almost frenzied. There is frequent defæcation of small quantities of fæces. In cases where the pain is due to uterine causes there is often some discharge from the vulva, which may be either mucoid or blood-stained. In the majority of cases the symptoms pass off in twelve to twenty-four hours, unless the pain is the preliminary indication of abortion. In the latter case the pain is more or less continuous until the abortion is completed.

Treatment.—The pain is best relieved by the administration of chloral hydrate ℥i., with or without the addition of a similar dose of bromide of potassium. This should be administered in a drench and dissolved in water. Morphia gr. iv.-vi. may also be given hypodermically. Alternatively to the above an alcoholic stimulant may be administered, the time-honoured dose of whisky often being effective in relieving symptoms. It is also well to administer a laxative or mild purgative, such as linseed oil or a small dose of Epsom salts. Hot cloths to the abdomen or a mild application of mustard plaster assist in the relief of pain. If the pain continues the chloral and bromide drench may be repeated in half-doses in six or eight hours. Hot enemata afford relief from the spasms.

Intussusception; Telescoping or Invagination of the Bowel in Cattle.—This is a fairly common cause of colic in cattle, and may be produced under similar circumstances as the same condition in horses. As in the horse the small intestine is most frequently affected, while sometimes the ileum may be intussuscepted through the ileo-cæcal valve, and at other times the cæcum itself may become invaginated.

Symptoms.—These commence with more or less severe abdominal pain, which usually lasts an hour or two, and then becomes subacute and continuous. In the early stages the animal may knock itself about, showing all the symptoms described in the spasmodic colic. After a few hours, however, when the subacute stage is reached, the violence of the symptoms abates and the patient becomes more or less dull and depressed, ruminating occasionally, and now and then taking a little food. During the first day or two, small quantities of fæces are passed, and this is followed by a period of several days during which there may be an absence of defæcation. At the end of that time a cast of the bowel may be passed consisting either of the cast of the mucous membrane or a portion of the telescoped bowel itself which may have become separated. The termination of this condition cannot be forecast. Some of the cases which appear more hopeful die, and some that look less hopeful recover. In the fatal cases the animal becomes very weak, breaks out into cold perspirations, and death is preceded by collapse.

Treatment.—It is impossible to reduce the telescoping of the bowel, and treatment is devoted entirely to the relief of pain. As in the case of spasmodic colic, chloral hydrate and potassium bromide are useful, and repeated mild laxatives should be given, but not violent purgatives. Large and frequent enemata should be given. After recovery from the acute symptoms a course of vegetable tonics should be given along with a demulcent diet.

Volvulus or Twist in Cattle.—This condition is very rare, but is occasionally met with involving the small intestines.

Symptoms set up are those of acute spasmodic colic and do not afford any basis for definite diagnosis. It is always fatal.

Treatment.—Owing to the impossibility of making a positive diagnosis, treatment is on the same lines as for spasmodic colic, and intussusception in cattle, otherwise slaughter is the only rational course to follow.

"Gut-tie" in Bullocks; also called "Pelvic Hernia."—This is a condition of strangulation of the intestine which is met with only in bullocks usually between one and two years old. It is brought about after castration, particularly when too much tension has been exerted on the spermatic cord, altering the normal relationships of the cord and peritoneum. It may also be due to leaving the cord too long. The cord may be partly retracted into the abdominal cavity and become attached to the internal inguinal ring or sometimes to the brim of a pelvis, thus forming a loop through which a portion of bowel may pass and become strangulated. In other cases the end of the cord becomes attached to the intestine itself and by subsequent contraction may cause obstruction and partial strangulation. Another asserted cause of the condition is the separation of a portion of the peritoneum adjoining the internal inguinal ring by undue traction during castration. This portion of the peritoneum may become ruptured, and a loop of bowel may pass through and become strangulated.

Symptoms.—Suddenly occurring spasmodic colic associated with the absence of fæces. Examination per rectum may reveal the strangulated portion at the brim of the pelvis varying in size. It is doughy in consistence and painful to manipulation. If the condition is not speedily relieved it is fatal.

Treatment.—An effort may first be made to reduce the strangulation by manipulation per rectum, or the offending loop may be broken down and the bowel thus released. Another way in which relief may sometimes be obtained is by galloping the animal down a steep hill; the forward jerking of the viscera by this process may bring about the release of the imprisoned bowel. If this fails, a surgical operation may be necessary. This is performed high up in the flank, the arm being inserted through the incision and the adhesion of the cord broken down or the peritoneal loop torn, thus releasing the imprisoned bowel.

Colic in Swine

Abdominal pain in pigs is fairly common, particularly in young pigs. It may result from tympany or flatulence, particularly in pigs that have been eating fermenting foods and damaged roots. It may also be caused by twist of the intestine, obstinate constipation, acute enteritis, intussusception, and peritonitis. It also occurs in uterine troubles.

Symptoms.—These depend somewhat on the cause. When due to tympany, there is a painful grunting, general uneasiness, standing up and lying down, sometimes eructations of gases, and sometimes vomiting. The abdomen is distended, and fæces are passed in very small quantities and frequently. Respiration is difficult owing to the pressure on the diaphragm. When the colic is due to strangulation or to volvulus the acute signs of pain last for several hours, the animal getting up and lying down, vomiting, grunting, and belching. Fæces are passed in small quantities at the outset of the attack, but there is soon a cessation of defæcation. When the condition is due to intussusception, the acute pains pass away after a few hours, becoming subacute and continuous, and the fæces are passed for a while and then are in abeyance. When the colic is due to volvulus or strangulation it is always fatal.

Treatment.—The treatment of colic in the pig is not very satisfactory, and if the animal is in prime condition it is well to consider the advisability of slaughter. If, however, treatment is decided upon, chloral hydrate or chlorodyne may be administered and the abdomen massaged and hot enemata given. Tympany may be relieved by the use of the trocar and cannula, which should be inserted on that side of the body where the distension is most prominent, sometimes right and sometimes left. Relief of the pain should be followed by a mild purgative, such as sodium sulphate or Epsom salts ℥iii.-iv., or castor oil or linseed oil ℥ii.-iv., according to the size of the pig.

Colic in Dogs

Acute abdominal pain is by no means infrequently met with in dogs, and may be due to a variety of causes, such as foreign bodies that have been swallowed (stones, skewers, needles, etc.), impaction of the bowels, twist of the bowels, intussusception, retention of the urine, and in bitches metritis. In puppies colicky pains are most frequently due to round worms (*Ascaris marginata*). Excessive tympany in the dog is very rare, as flatulence is usually relieved by eructations or by the passage of flatus.

Symptoms.—Acute abdominal pain in the dog is shown by cramping pains of the abdominal wall and an expression of distress and wistfulness, vomiting, and the passage of fæces and urine spasmodically. Temperature may or may not be elevated. The patient cries on being handled or picked up, and any palpation of the abdominal wall is objected to by the patient. Skewers or needles may work their way from the stomach through the abdominal wall or between the last ribs. Their point of exit is first shown by a circumscribed swelling, which develops into an abscess and bursts, revealing the point of the offending body. In puppies where the colic is due to worms screaming fits may occur, the attacks occurring at intervals and being most distressing.

Treatment.—The most rapid relief is afforded by the hypodermic injection of morphia in doses from gr. $\frac{1}{4}$ to gr. ii. according to the size of the dog. This will usually afford relief in a few minutes, lasting from twelve to twenty-four hours. At the same time it relaxes the abdominal muscles and facilitates the subsequent examination of the abdomen, with a view to a diagnosis of the cause of the condition. Good results also are obtained by the use of syrup of chloral mm. xv. and potassium bromide gr. xv., or of chlorodyne from mm. ii.-x. and enemata. Hot cloths sprinkled with turpentine afford considerable relief from the pain. Further treatment must depend entirely upon the diagnosis of the cause of the condition. Skewers or needles should be removed surgically (see p. 739). For the treatment of worms in puppies see p. 323.

Obstruction of the Bowels in Dogs and Cats.—This condition is commonly due to foreign bodies, as stones, corks, small balls, coins, and sometimes to bones.

Symptoms.—The first thing observed is repeated vomiting, which becomes very persistent. This is sometimes associated with dribbling of saliva. There may be a little appetite in the early stages, but when the obstruction is complete anorexia supervenes. Fæces may be passed for a while. Repeated vomiting leads to great exhaustion and rapid wasting, and the patient has a very distressed expression of countenance. If the obstruction has reached the large intestines vomition is less constant and sometimes progressive paralysis of the hind legs supervenes. Palpation of the abdomen may reveal the presence of the foreign body, care being taken to distinguish it from normal ingesta or in cats from the kidneys. Digital exploration per rectum may also help. In cases where there is any doubt in diagnosis the patient may be submitted to the X-rays.

Treatment.—If the obstruction is due to impacted food materials an oily purgative may be given together with frequent enemata of glycerine and water, or oil or soap solution, and the abdomen kneaded to soften the ingesta. Hot cloths to the abdomen may afford considerable relief. If a foreign body is diagnosed as the cause of the obstruction a surgical operation is necessary. In cases where a stone or similar foreign body is known to have been swallowed and has not passed the stomach, relief is best obtained by giving the dog a feed of rather large pieces of meat, which he will probably bolt without mastication. In ten to fifteen minutes apomorphine gr. $\frac{1}{20}$th to $\frac{1}{12}$th should be given hypodermically. This causes rapid vomition and the foreign body is usually expelled along with the food contents of the stomach.

Intussusception in the Dog.—As in the case of the larger animals intussusception in the dog most frequently involves the small intestines, the jejunum in particular. The invaginated part varies in length from an inch or two upwards, the common length being three or four inches. A fairly common condition also is the passage of the ileum through the ileo-cæcal valve into the large intestine, and sometimes the intussuscepted part may be so extensive as to protrude through the rectum and anus and to be mistaken for prolapsed rectum.

The causes of the condition are varied; it will sometimes occur from sudden exaggerated movement such as falling off a table or chair; sometimes it appears to be brought about by worms causing irregular peristalsis and continuous straining. Occasionally intussusceptions may occur simultaneously in several of a litter of puppies in what might almost be described as an "outbreak." In such cases one cannot even hazard a suggestion as to the cause.

Symptoms.—The symptoms appear to correspond largely with those of obstruction of the intestines due to foreign body. There is persistent vomiting and almost continuous straining with the passage of only small quantities of fæces to begin with, and later the absence of fæces. On manipulation through the abdominal wall the intussusception can frequently be felt somewhat resembling a firm sausage, and is somewhat painful on pressure. When the ileum is invaginated it may protrude through the rectum, and must be distinguished from prolapse of the rectum. This can be done by inserting the finger between the everted bowel and the anal rim and attempting to pass it into the rectum. This is easy in the case of intussuscepted ileum, but when the everted part consists of rectum the skin at the anal rim is usually directly continuous with the everted bowel, and the finger will then not pass in. It is important to differentiate because in the latter case the rectum can be returned easily with a good prospect of ultimate success, while the intussusception is far more serious and more difficult to reduce. Affected dogs rapidly become seriously ill, and if the intussusception is not quickly reduced it is likely to become gangrenous and terminate fatally in a few days.

Treatment.—If the case is seen early a narcotic should be given and an attempt made to reduce the intussusception. A method suggested by Livesey consists in slowly distending the bowel with a stream of warm soapy water per rectum. Several pints may be injected slowly and with some force in order to get it past the ileo-cæcal valve, massaging the abdomen occasionally and care being taken not to damage the rectum by over-distension. He suggests that this method may actually reduce the intussusception, but is only of service if undertaken early. It would not reduce ileo-cæcal intussusception. A more certain method if the case is seen early is to perform laparotomy and draw out the telescoped portion with great care. If this cannot be accomplished or if the intestinal wall is badly injured enterostomy must be performed (see p. 739). In cases where the ileum is involved treatment is less likely to be successful. It occasionally happens that an intussuscepted part becomes spontaneously separated and passed per rectum. When the intussusception is reduced the patient should be kept under an anodyne to reduce peristalsis and further straining. For this purpose chlorodyne mm. ii.-x. is probably the best, administered three times daily in petroleum emulsion ʒi.-iii., or paraffinum liquidum to lubricate the intestine mechanically and control the constipating effects of the chlorodyne. The subsequent diet for some days should consist solely of liquid foods, such as milk with egg, or beef-tea or barley water. Absolute quietness must be enforced.

Volvulus in the Dog.—This condition is exceedingly rare but may occasionally be met with. The small intestine is usually affected and the cause is unknown. Strangulation of the intestine may sometimes be due to the presence of Meckel's diverticulum and more frequently to hernia.

Symptoms.—The symptoms develop very rapidly, there is acute abdominal pain and frequent vomiting and great distress. The dog has a very anxious expression of countenance and soon becomes almost collapsed. On manipulating the abdomen the muscles are found tense and palpation is painful and is objected to. Positive diagnosis is, however, difficult and can only be established by an exploratory laparotomy.

Treatment.—No medicinal measures are of any service, and relief can only be successfully afforded by performing laparotomy and releasing the twist or the strangulation.

PARASITES OF THE ALIMENTARY TRACT

Parasites of the Pharynx

Horse Leeches (Hæmopis sanguisuga).—Leeches may be met with in the pharynx of horses and cattle, having been taken in with the drinking-water. They are temporary parasites with soft, elongated, segmented bodies, and furnished with two suckers. By their oral suckers they may attach themselves in various situations, including the pharyngeal mucous membrane, where they suck blood, swelling up to a considerable size and producing local inflammation. Their presence is indicated by hæmorrhage from one or both nostrils, and if they are very numerous, hæmorrhage may be very severe and anæmia may be set up.

Treatment consists in irrigating the nostrils and pharynx with salt solutions, vinegar, ammonia, or turpentine. The parasites may be seen by the use of a mouth speculum on drawing the tongue forward, and in these cases may be removed by forceps or by cutting across with the scissors.

Prevention consists in the filtering of drinking-water which may be infected, or the provision of fine-net muzzles.

Parasites of the Æsophagus

Several parasites have been found in the œsophageal mucous membrane and in the muscular tissue of the gullet. They include :

Spiroptera sanguinolenta in dogs ;
Gongylonema scutatum in horse, ox, sheep, goat, and pig ;
Larvæ of *Hypoderma lineata* in cattle ;
Psorosperms (*Balbiania gigantea*) in ruminants and horses (see Parasites of the Connective Tissues, p. 427).

Spiroptera sanguinolenta.—*Spiroptera sanguinolenta* is a blood-red nematode worm from two to four inches long. It produces submucous tumours up to the size of hazel-nuts containing a number of parasites.

They are chiefly met with in dogs in Italy, Algeria, Tunis, China, Japan, India, and Brazil. Dogs become infected with this parasite by eating cockroaches which harbour the intermediate stage of the worm, the larva being liberated in the stomach and migrating forwards into the œsophagus.

Symptoms.—In cases of severe infestation these parasites set up general indisposition, anorexia, painful swallowing, and frequent vomiting and emaciation, and may prove fatal.

Treatment.—There is no curative treatment, but sedatives such as chlorodyne and bromides, together with generous feeding, materially relieve the symptoms.

Prevention consists in the extermination of cockroaches.

Spiroptera scutata (*Gongylonema scutatum*).—These are long, thread-like worms which occur laced through the mucous membrane of the œsophagus, and sometimes of the rumen and reticulum. The male is about 2 inches long, and the female from 5 to 6 inches long. It has been met with in Polish and Hungarian oxen, and in American cattle and sheep. It also occurs in cattle in tropical Africa. It is not known to give rise to any symptoms.

Parasites in the Stomach of the Horse

Larvæ of the Gastrophilus equi; Œstrus equi; Stomach bots (p. 495).—The *larvæ of the Gastrophilus equi*, which is a dipterous insect, pass a considerable portion of their existence in the stomach of the horse. They are called horse bots and are only met with in horses that have been out at pasture. The flies deposit their eggs in the summer and early autumn on the hair generally of the legs and shoulders. The eggs are very small, elongated, and yellow, and adhere very firmly by means of a kind of cement. They hatch out in a few days and liberate tiny larvæ or maggots, which are licked off by the horse and taken in by the mouth. On reaching the stomach they usually become attached to the cuticular or cardiac end of the stomach, and there they remain until the following spring or early summer. By this time they have become mature and detach themselves, and are passed out with the contents of the stomach and intestines along with the dung. Under favourable conditions they develop into the chrysalis, and in about a month the adult fly is hatched out. The fly is a little larger than the ordinary house-fly, about half an inch long, and is yellowish-brown in colour. (For further details of the fly and the larvæ see chapter on Ectozoa, p. 495.) Other larvæ which may also be found in the stomach of the horse include:

Larvæ of the Gastrophilus nasalis, which may be met with in the pyloric end of the stomach and the duodenum. The adult fly, which is brown, deposits its eggs on the nostrils of the host, from which the larvæ drop into the food and are ingested.

Larvæ of the Gastrophilus hæmorrhoidalis.—The larvæ of this fly are somewhat smaller than those of the *Gastrophilus equi*. The eggs are deposited mainly on the lips and sometimes on the anterior part of the body of the horse. The name is derived from the fact that on their way out from the stomach the larvæ become attached to the rectal mucous membrane for some time before being expelled.

Larvae of the Gastrophilus pecorum.—These larvæ are dark brown in colour and are found in the stomach and duodenum, and, like the *G. hæmorrhoidalis*, remain in the rectum for a time during passage out of the body. They are found chiefly in Hungarian and Russian horses. The eggs are deposited on the lips and nostrils, as in the case of the *Gastrophilus nasalis*.

Gastrophilus flavipes.—The larvæ of this parasite are met with in the stomach of the donkey, particularly in Southern Europe.

Symptoms of Bots.—Unless these parasites are present in very large numbers they apparently cause no ill-effects, and their presence may never be suspected until larvæ are observed being passed with the dung or remaining attached to the rim of the anus. In a few cases they may give rise to indigestion and unthriftiness. On rare occasions, however, they have been known to perforate the stomach wall and to set up peritonitis. In other cases, when present in large numbers, they may have been thrown off in bulk, and have actually caused obstruction of the small intestines with corresponding symptoms. In the case of the parasites which may remain in the rectum for a time some irritation may be set up with frequent defæcation, tenesmus, and rubbing of the tail.

Treatment.—It is often very difficult to cause the expulsion of these parasites, as they are very resistant to most parasiticides. The most effective agent appears to be carbon disulphide, which may be administered in gelatin capsules in doses of ʒiii. or ʒiv. to adults and half those doses for foals. This should be repeated three or four times at intervals of two hours. The animal should have been kept fasting the previous day, and treatment should commence in the early morning. This should be followed by a dose of oil and turpentine. The effect of this large dose of carbon disulphide may cause partial narcosis of the patient; he may reel about and become unsteady. In very susceptible but exceptional cases death has followed. Another treatment consists in the administration of tincture of iodine ℥ss. in capsule for four or five successive mornings about two hours before feeding, the last dose to be followed by a draught of oil and turpentine as before. Parasites in the rectum may be removed by hand, and also by the injection of soapy solution with or without turpentine added, or of carbon disulphide in oil. Any larvæ expelled should be destroyed, otherwise they are likely to hatch out, giving rise to a fresh crop of flies. Eggs seen on the limbs of the horse should be removed by clipping and the hair burned. They adhere so firmly that any other attempt at removing them fails.

Ascarides (*Ascaris megalocephala*) are sometimes met with in the stomach of the horse. Their normal habitat, however, is the small intestines, but they may migrate forwards into the stomach. In this situation, unless present in large numbers, they rarely give rise to any symptoms.

Spiroptera megastoma, Spiroptera microstoma.—These thread-like worms are sometimes met with in small tumours of the mucous membrane of the stomach in horses and donkeys. They rarely give rise to any symptoms which can be detected during life, their presence being only discovered on post-mortem examination.

Trichostrongylus axei (*Strongylus axei*).—This parasite is also occasionally met with in small nodules in the wall of the horse's stomach. Like the spiroptera, it gives rise to no clinical symptoms, and is only discovered on post-mortem examination.

Parasites of the Stomach of Ruminants

Strongylosis.—Ruminants are subject to infestation of the abomasum and small intestines by a number of very small thread-worms or strongyles. The condition set up is either parasitic gastritis or gastro-enteritis according to the parasites present.

The parasites mainly responsible are:

Parasite.	Synonym.	Length.	Habitat.	Host.
Hæmonchus contortus	Str. contortus	m. ½″-¾″, f. 1″	Abomasum and ? intestine	Cattle, sheep, goat.
Ostertagia circumcincta	,, cervicornis	m. ⅓″, f. ½″	Abomasum and intestines	Sheep, goats.
,, ostertagi .	,, convolutus	m. ¼″-⅓″, f. ⅓″-½″	Abomasum	Cattle.
Cooperia oncophora .	,, ventricosus	m. ⅓″, f. ⅓″-½″	Intestines	Calves, occasionally sheep and goats.
Nematodirus filicollis .	,, filicollis	m. ½″-⅗″, f. ⅘″	Intestines	Cattle, sheep, goat.
Trichostrongylus axei .	,, gracilis	m. ⅛″-⅙″, f. ⅕″	Abomasum	Cattle, sheep, goat.

The worms are met with particularly in low-lying and marshy or swampy districts, and affect young animals more seriously than adults. The conditions favourable to the existence and development of these parasites are also favourable to the development of various others. Hence young cattle affected with parasitic gastritis often harbour *Strongylus micrurus* (Hoose worms) in the trachea and bronchial tubes; while sheep are frequently infected with *Strongylus filaria* (Hoose worms) and *Strongylus rufescens* in the bronchi and in the lungs respectively, and also with *Trichocephalus affinis* (whip-worms) and various tapeworms in the cæcum and colon.

Infection generally takes place during pasturing on land which has been previously pastured by affected animals. In districts where the disease is prevalent a very large proportion of the adult cattle and sheep harbour parasites apparently without suffering any deleterious effects. Such animals, however, are constantly passing eggs and so contaminating pastures, and young animals of the same species grazing after them are likely to become seriously affected. In this way outbreaks may frequently assume the proportions of an enzoötic, particularly in lambs.

Symptoms.—These are mostly observed in the wet season and consist, in the first place, of general unthriftiness and great loss of condition, with irregular bowels.

In lambs and first-year sheep diarrhœa may be set up which is very persistent, leading to intense anæmia. Lambs are frequently found dead after having been ill only a day or so. As it may affect a number, it not infrequently gives rise to suspicions of foul play or poisoning. This is especially so in cases of sudden intensive infestation. On examining the droppings in the pastures in such cases they are found to vary considerably, sometimes diarrhœic, and others "formed" and followed by a quantity of jelly-like mucus, which may be blood-streaked.

In cattle, however, the condition usually runs a more protracted course, and animals affected are frequently called "wasters" or "piners." Young cattle develop a kind of pernicious anæmia, with a fœtid, watery, and persistent diarrhœa. They become emaciated and hide-bound, thirsty and empty-looking, with arched back and "tucked-up" abdomen and hollow flanks. Temperature becomes subnormal and death occurs from exhaustion.

Diagnosis of the condition during life is not always easy. Both parasites and eggs may be passed with the fæces, but unless in huge numbers it cannot be assumed that they are always responsible for the symptoms. It is a question of numerical invasion. In cattle it may readily be confused with tuberculosis or with Johne's disease. In the case of sheep, if there is not a dead animal available for post-mortem examination it is better to slaughter one that appears to be seriously ill and make a post-mortem examination to establish a definite diagnosis.

Post-mortem Appearances.—In acute cases the carcase may still remain in good condition and well nourished, but in the majority of cases one finds indications of anæmia, emaciation, and general lack of nutrition. The chief features are met with in the abomasum and the first portion of the small intestines. The mucous membrane in acute cases, due to sudden intensive invasion, may be somewhat red and inflamed, but in other cases the membrane is pallid, rather thickened and catarrhal. The contents of the fourth stomach are very liquid and frequently green in colour. The liquid contents contain large numbers of the small parasites, which, however, are usually not discerned by the naked eye. They can readily be demonstrated, however, by putting just a small quantity of the stomach contents by means of a needle into a flat-bottomed glass vessel with water, shaking and allowing to settle, and then examining by the naked eye with the glass placed over a dark background, such as one's sleeve; or the examination may be made with a hand lens or under low power of the microscope. If necessary, individual parasites can be picked out and placed on a glass slide for closer microscopic examination and identification. These parasites can only be regarded as serious and the probable cause of death if they are present in considerable numbers.

Treatment.—The curative treatment of acute parasitic gastritis in ruminants is not very successful, and the same applies to chronic cases which have been allowed to go on for some time and in which the mucous membrane has become denuded and the patient very emaciated.

In cattle, if taken in good time, a solution of lysol ʒiv. in water Oi. may be given fasting, and repeated in half that dose twice daily for several days. If the animal is tractable this is best administered by means of the stomach-tube in order to avoid producing stomatitis. On the other hand, if it is given too diluted it is almost useless. Another useful combination consists of ol. tereb. ʒii., acid. carbol. ʒii., to be given in milk Oi. This may be repeated in half-doses in several successive days. If drenching is impossible or objected to, a useful combination consists of pulv. arsenic gr. x., antim. pot. tart. ʒi., with ferri sulph. ʒiv. This to be given daily for a week in the food. Other agents used with varying success are copper sulphate (ʒi.-ʒii.), creosote, kamala, and areca nut. The diarrhœa may be controlled with intestinal astringents, such as pulv. opii ʒss., pulv. catechu ℥ss., and pulv. creta prep. ℥i., to be given twice a day in starch gruel or in the food. The animal should also be brought up off the pasture and fed with easily digested dry diet, which may with advantage include cotton-cake.

In outbreaks **in sheep** the animals should be removed from the infested pasture and, if possible, placed in a dry fold or, alternatively, should be folded upon roots so that they can be constantly moved on from day to day. The object of this is to reduce further infestation of the lambs and also to reduce the contamination of the pasture. Medicinally, vermicides may be administered with the trough foods, and good results are obtained by the combination of copper sulphate ʒss. with powdered white arsenic gr. v., to be given once daily for a week. If there is no objection to drenching the sheep one of the best methods of treatment is the administration of 1 per cent solution of copper sulphate in doses of 3 to 4 ounces to adult sheep and from 1 to $1\frac{1}{2}$ ounces to lambs under one year old. Another mixture recommended consists of ol. tereb. ʒii., acid carbol. mm. x., chloroform mm. v., administered either in a few ounces of milk or soap solution. Other parasiticides used include thymol, areca nut, oil of cade, lysol, creosote, and beta naphthol. These vermicides may be followed by a course of tonics, such as gentian and iron in the trough food. A lump of rock-salt in the trough is useful for both cattle and sheep.

Preventive measures should always be adopted, and one of the first principles to note is that lambs should never be grazed after adult sheep. If possible a fresh pasture should be retained each season solely for the lambs. In badly infested districts it is a very good plan to dose all the sheep and lambs twice or three times every season with copper sulphate solution before symptoms are developed, beginning July and repeating in August and September. This practice no doubt checks the development of the disease and is responsible for saving a considerable number of lambs each year. Pastures known to be infested should, if possible, be kept vacant of ruminants for at least a year[1] and should be dressed generously with salt or lime. Since horses are not susceptible to these parasites they may be turned out on to the pastures. When such a thing is possible it is a good plan to turn infested pastures under the plough.

Amphistomum conicum.—This is a small trematode found in the rumen of cattle and sheep. It has an oval or conical body about $\frac{2}{5}$ inch long and is reddish white in colour. It is met with especially in the spring, and as a rule it gives rise to no appreciable symptoms. It has, however, been regarded as a cause of fatal outbreaks of disease in cattle in Australia.

Parasites of the Stomach of Swine

The following round worms are met with in the stomach of pigs:

1. *Spiroptera strongylina.*
2. *Gnathostomum hispidum.*
3. *Simondsia paradoxa.*
4. *Strongylus rubidus.*
5. *Ascaris suis.*

(1) **Spiroptera strongylina.**—This is a small white worm; length: male up to $\frac{2}{3}$ inch, female up to $\frac{4}{5}$ inch. It is met with in the mucous membrane of the stomach and the submucous tissues, where it may form small tumours, and in some instances may set up severe gastritis

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[1] As Theiler states, this is often difficult or impossible. His alternative suggestion is to have the larvæ picked up by sheep, and to kill them in the stomach of the sheep. Since the parasitic worm requires one month to reach sexual maturity before it again lays eggs, the dosing must take place within a month, and must be repeated during the months of the rainy season. After the lapse of one year the mature larvæ which have not reached their host have practically all died, and during this time no eggs have been scattered about, so that after the one year a farm should be clean of wire-worms.

with fatal results. In some parts of the U.S.A., such as Chicago and Kansas City, 60-80 per cent of the pigs are said to be infested. It is not recorded as occurring in the British Isles. The exact nature of the infection is not discovered until post-mortem examination is made.

(2) **Gnathostomum hispidum.**—This is a thread-worm up to $1\frac{1}{2}$ inches in length. It attaches itself to the mucous membrane of the stomach and occasionally may bore into it, the posterior extremity projecting into the cavity of the stomach. It is not a very common parasite, but when present in large numbers may set up acute gastritis with vomiting and indigestion and general wasting. It occurs in Africa, Asia, Europe (Hungary). It has not been recorded as occurring in the British Isles.

(3) **Simondsia paradoxa** is a very rare parasite. The females are found in cysts in the stomach wall and are from 1 to $1\frac{1}{2}$ inches in length. The males are found in the cavity of the stomach.

(4) **Strongylus rubidus.**—This is a very small worm, the male being $\frac{1}{4}$ inch long, the female $\frac{1}{3}$ inch. It occasionally occurs in England without causing serious inconvenience. In America, however, and in certain parts of the Continent of Europe it occurs more extensively and is sometimes responsible for outbreaks of parasitic gastritis resulting in great anæmia, wasting, and death. The eggs of the parasites can be demonstrated in the fæces on microscopic examination.

(5) **Ascaris suis.**—This is a cylindrical worm whose normal habitat is the small intestines. It occasionally migrates forward, however, into the stomach, and may then cause vomiting and convulsions.

Treatment for Gastric Worms in Swine.—In cases where the positive diagnosis is made the animals may be given a course of sulphate of iron ʒi.-ʒii. daily for a week or ten days. This may then be followed by calomel ʒss. with jalap ʒiii.-ʒiv.

Preventive measures consist in careful disposal of the fæces, which invariably contain numerous eggs and are responsible for further infection.

Parasites of the Stomach in Dogs

The following parasites may be met with in the stomach of dogs :

1. *Spiroptera sanguinolenta.*
2. *Gnathostomum spinigerum.*
3. *Belascaris marginata.*
4. Various *Tæniæ.*
5. Larvæ of *Gastrophilus equi.*

(1) **Spiroptera sanguinolenta.**—This worm is also found in the œsophagus and has already been described on p. 312. In the stomach it may be found in small tumours which may attain the size of a pigeon's egg, but are rarely numerous. They may cause persistent vomiting with great loss of condition and anæmia, and may ultimately prove fatal. In rare cases they cause rupture in the stomach wall. These parasites have also been met with in other parts of the body, such as the lungs and lymphatic glands. For treatment and preventive measures see *ante.*

(2) **Gnathostomum spinigerum.**—This is a round worm which may be met with in both the dog and cat. The male is about $\frac{1}{5}$ inch long and female about $\frac{2}{5}$ inch. It is met with in small tumour-like elevations under the mucous membrane, and appears rarely to cause any symptoms.

(3) **Belascaris marginata** and (4) **Various Tæniæ.**—Ascarides and tapeworms may be met with in the stomach of dogs, having migrated forward from their normal habitat, the intestines. In this situation they may give rise to persistent vomiting, particularly in young dogs. Some of the worms may be ejected with the vomit.

(5) **Larvæ of Gastrophilus equi.**—These maggots have occasionally been met with in the stomach of the dog, but appear to cause no inconvenience. They have probably been swallowed when the animal has been fed on horse flesh or horse's stomach, or they may have been picked up with horse manure and have attached themselves to the gastric mucous membrane.

Parasites in the Stomach of Cats

The following worms may be met with in the stomach of cats :

1. *Ollulanus Tricuspis.*
2. *Gnathostomum spinigerum.*
3. *Belascaris cati.*
4. Various tapeworms.

(1) **Ollulanus tricuspis.**—This is a very small worm up to $\frac{1}{25}$ inch in length, and therefore microscopic in size. The adult worms are found in the gastric mucous membrane, and others pass on into the intestines. In the stomach they may give rise to gastritis, causing vomiting and wasting. From the stomach embryos which are deposited by the adults migrate into the various tissues, such as diaphragm, lungs, and liver, and become encapsuled in small cysts, setting up the condition known as ollulaniasis. Other embryos are passed out with the fæces, and are ingested by rats and mice, where a similar life-history is passed through. Cats become infected by ingesting affected rats and mice.

(2) **Gnathostomum spinigerum** (see Gastric Parasites in Dogs).

(3) **Belascaris cati** and (4) **Various Tapeworms.**—These may be met with in the stomach of cats, and, as in the case of the dog, they have

migrated forward from the intestines. They set up more or less persistent vomiting until they are expelled with the vomit (see Intestinal Parasites).

Intestinal Parasites of the Horse

Nematodes.—Ascaris Megalocephala.—This is the commonest intestinal parasite of the horse. It is a large yellowish-white worm, rather stiff or turgid, up to a foot in length, and may be as thick as an average lead pencil. It has a well-marked head possessing three lips. Ascarides are usually met with in the first portion of the small intestines, and may be present in large numbers. In rare cases, indeed, they may be sufficiently numerous to cause a complete obstruction of the bowel. In other cases the parasites pass forward and may be found in the stomach.

Symptoms.—When the parasites are only a few in number they rarely cause any symptoms. When they become numerous they may set up a superficial catarrhal enteritis with loss of condition and some irregularities of the bowel. Frequently a small quantity of fluid is expelled from the anus after defæcation.

The appetite is variable, but is rarely completely lost. Ascarides may cause intermittent colic, and sometimes complete obstruction and even rupture of the bowel. It is suggested that they are sometimes responsible for irregular peristalsis such as may bring about volvulus or twist. This, if it ever occurs, however, must be rare. Brain symptoms are sometimes attributed to ascarides in the horse, and these are due to certain poisonous products of the parasites which are absorbed, setting up convulsions in some cases, muscular spasms suggestive of tetanus in other cases, and occasionally proving fatal.

Definite diagnosis may be made either by the presence of the parasites in the fæces, or the discovery of their eggs, which are round or ovoid, by microscopic examination.

Treatment.—These worms are comparatively easily expelled. A very reliable and effective combination consists of pulv. arsenic alb. gr. v., antim. pot. tart. ʒi., ferri sulph. ʒii., pulv. gentian ʒii. This powder should be given in a small mash one or two hours before the first meal every morning for six days, and should then be followed on the seventh day by a simple purgative such as an aloes ball, or a combination of aloes and calomel, or linseed oil Oiss., to which may be added ol. tereb. ℥ii. The latter may be effective alone without the previous course of vermicides. Other useful agents consist of santonin gr. xxx. or xl., or thymol gr. xl.-ʒi. All parasites expelled should be destroyed by burning, and not allowed to be carried away with the manure.

Strongylosis—Sclerostomiasis.—There is a considerable number of strongylidæ met with in the horse. They involve the large intestines, especially the cæcum and colon. They were formerly collected into two groups. The larger ones were called *Strongylus armatus major* (with their larval stages, *Str. armatus minor*, in aneurisms of the great mesenteric artery). The smaller worms were all called *Str. tetracanthus*. Further investigation has shown that a considerable number of worms is represented.

They consist of three principal genera, namely:

(*a*) *Strongylus* (or *Sclerostomum*);
(*b*) *Triodontophorus*; and
(*c*) *Cylichnostomum*.

(*a*) The genus *Strongylus* (or *Sclerostomum*) is represented by three worms, viz.:

Str. equinus, which is about 1½ to 2 inches long, and is the largest. It inhabits the lumen of the bowel, and its larvæ are in the bowel wall.

Str. edentatus, somewhat smaller than the former with similar habitats.

Str. vulgaris.—This is the smallest, the adult being about ⅔ to 1 inch long, and the larvæ are found in the large mesenteric arteries causing aneurism.

(*b*) The genus *Triodontophorus* comprises three worms, viz.:

T. serratus, about ¾ inch long.
T. minor, about ½ inch long.
T. intermedius.

(*c*) The genus *Cylichnostomum* consists of 16-17 worms varying in size from ⅓ inch to 1 inch. The most important one is *C. tetracanthum* (*Scleros tetracanthum*, *Str. tetracanthus*, or *Trichonema arcuata*), which is a reddish worm from ⅓ to ½ inch long, and often adheres to one's arm when back-raking infested horses.

Some of the strongylidæ are very vicious parasites, and cause considerable injury to the bowel wall by biting and blood-sucking; but more serious effects are caused by their larval stages within the bowel wall during their passage through the intestinal wall, when they are passing from the aneurism in the roof of the abdominal cavity into the lumen of the bowel (*Str. vulgaris*).

Horses become infested with these parasites when out at grass and usually in low-lying or marshy pastures. Infection is also possible by means of hay made from such pastures.

Symptoms.—The symptoms are principally met with in the autumn and winter, or sometimes in the early spring. The effects are often very serious, particularly in young horses. The symptoms first set up are those of loss of condition and loss of flesh with progressive anæmia, pendulous abdomen, hollow flanks, staring coat. The bowels become irregular with a tendency to diarrhœa, which may become profuse and very offensive. The mucous membranes become pale and watery, and sometimes in bad infestations somewhat yellowish, probably due to hæmolysis caused by toxic products of the parasites. The appetite is variable: sometimes well maintained, and in other cases the patient refuses food for varying periods. The temperature is irregular, but rarely very high. On examination of the fæces parasites, small red worms, may be discovered frequently, and on back-raking parasites may be discovered in the rectum and stick to one's arm. In bad cases death occurs as the result of exhaustion and anæmia from the diarrhœa and probably hæmolysis. In very acute cases death may result in from several days to a week after the onset of symptoms. At other times a case may be protracted and last for weeks, occasionally terminating in recovery, but more frequently terminating fatally. In protracted cases death is preceded by very great debility, and the horse may be unable to stand, giving rise to a diagnosis of paralysis. (For the symptoms of verminous aneurism see p. 304.)

Post-mortem Appearances.—The parasites may be found free in the large intestine, while others may be found present in the intestinal wall in small tumour-like nodules about the size of peppercorns. These can be best seen by washing the portion of the bowel and holding it up to the light, where it gives the appearance as though almost riddled by shot. On closer examination some of the nodules may show small punctures through which the worms have escaped into the bowel. In other cases there may be patches of deep-seated inflammation of varying size involving the cæcum and colon. The carcase shows all the phenomena of anæmia, prominent amongst which are some dropsy of the peritoneum and myxomatous degeneration of the renal fat.

Treatment.—When the parasites are present in the intestinal wall treatment is very unsatisfactory. If they are limited to the lumen of the intestine, as in the case of the cylichnostomes and adult strongyles, they may be more easily dealt with. The horse should be taken up from the pasture and put into a comfortable stall, and be fed on highly nutritious dry foods. The inclusion of beetroot in the food has been strongly recommended, and of itself may cause the expulsion of numbers of intestinal parasites. Numerous agents have been tried with varying results. The best results are afforded by oil of chenopodium ʒii.-ʒiv. given with, or followed by, a quart of linseed oil, the patient having been previously fasted twenty-four to thirty hours. Good results may also be obtained with the combination of thymol gr. xxx.-xl. dissolved in rectified spirits ℥ss. with oil of chenopodium ʒi., administered in ol. olivæ ℥ii. twice daily for a week, and followed by a purgative dose of linseed oil. Similar measures may be adopted as in the case of ascarides, the combination of arsenic, antimony, and iron being found very useful in many cases. Thymol ʒi. in bolus, or santonin gr. xl. daily for a week, followed by a purgative dose of calomel and aloes, sometimes gives good results. Pure paraffin oil Oss. administered undiluted as a drench two or three times within a week has also been found effective. Persistent diarrhœa should be controlled or checked by intestinal astringents such as opium or chlorodyne and catechu.

Preventive Measures.—These should be adopted in localities where the parasites abound. They consist in attention to the pastures, which should be well drained and generously dressed with lime or salt and left free from horses two winters and one summer. Alternatively, the pastures may be ploughed up. In indigenous districts it is a good plan to dose horses with parasiticides periodically without waiting for symptoms to be shown. The powder already recommended, consisting of arsenic and antimony, etc., may be given daily for a week and repeated every two or three months. The following made up in bolus is also good:

Hydrarg. subchlor.
Antim. pot. tart. āā ʒi.
Ferri sulph.
P. aloes āā ʒii.
Saponis moll. q.s.
Ft. bolus.

One to be given every two months after 12 or 24 hours' fasting.

Oxyuriasis.—This is the name given to infestation with *Oxyuris curvula* and *Oxyuris mastigodes*, two parasites which closely resemble each other, the first one being 1½-2½ inches long and the other often two or three times this length. They are thick at one end and taper away to the tail end, and this shape has given rise to the name *false whip-worm*. These worms infest the large intestines, particularly the colon and rectum. Their presence in the latter situation has given rise to the name *seat-worm.*

Symptoms.—Members of the class of oxyuris only rarely give rise to symptoms of practical importance. Frequently their presence is not even suspected until some are seen passed out with the fæces or a single worm may be seen protruding from the anus. There is sometimes a yellowish white crust round the anus which consists largely of dried mucus and eggs of the parasite. Their presence may give rise to irritation of the anus (pruritus ani) and straining after passing fæces (tenesmus), and sometimes to persistent rubbing of the tail. The latter symptom, however, may also be set up as the result of acariasis, a form of mange.

Treatment.—The parasites are best removed by a course of arsenic, antimony tartrate, and iron, followed by a dose of physic. A draught of linseed oil with turpentine is often effective. Rectal injections of vermicides are often resorted to, and these are probably only effective in the case of parasites in the small colon and the rectum. Agents used for this purpose consist of strong salt solution or infusion of quassia (1-20), infusion of tobacco (1-40), turpentine in soap solution, and lysol 1 per cent. Before these injections are administered the horse should be back-raked, and Oi. of the solution should be injected at about blood-heat. This should be repeated daily for a week.

As no intermediate host is apparently necessary for the development of these worms, infestation may take place by the contamination of fodder with discharges from an affected animal. Similarly eggs may be directly transferred by grooming-utensils, particularly the sponge and rubber, from the anus to the lips. Steps which are therefore obvious should be taken to prevent this transference.

Cestodes or Tapeworms in the Horse.—There are only three tapeworms harboured by the horse. They belong to the class *Anoplocephalinæ*, unarmed tapeworms, and their life-history is unknown. The commonest is the *Anoplocephala perfoliata* (*Tænia perfoliata*), which is from 1 to 2 inches long. The smallest, which is the *Anoplocephala mamillana* (*T. mamillana*), is from ½ to 1 inch long, and is next in frequency. The remaining one is *Anoplocephala plicata* (*T plicata*), which is 1-3 inches long. The common habitat for the first two of these worms is the cæcum and sometimes the colon, rarely in the small intestine. The other inhabits the small intestines.

Symptoms.—These tapeworms in the horse rarely give rise to any symptoms by which they can be diagnosed during life. When present in large numbers they may be responsible for general unthriftiness and anæmia, and one or two cases of peritonitis have been recorded due to perforation of the intestines by tapeworms.

Treatment.—As these tapeworms are unarmed they are only attached to the mucous membrane by means of their suckers and consequently are comparatively easily expelled. An effective procedure is to administer a course of freshly-ground areca nut ʒiv. with a dram of antimony tartrate once daily for a week and follow with a dose of physic or a draught of linseed oil and turpentine.

Trematodes of the Horse.—The trematodes met with in the horse include:

1. **Amphistomum collinsi** (**Pseudodiscus collinsi**).—These are short thick trematodes which are occasionally found in immense numbers in the large intestines of horses in India, where they give rise to a condition which has been called *masuri*. The parasite is about a quarter of an inch long and half as wide, and is brick-red in colour.

The symptoms it sets up are those of indigestion, unthriftiness, and sometimes enteritis. Worms and eggs may be found in the fæces.

Treatment consists in the administration of repeated doses of filix mas, which should be followed by a dose of physic. Prevention consists in the avoidance of infected water and pastures. In districts where the disease is common all the water should be first filtered. Pastures might be dressed with salt.

2. **Gastrodiscus ægyptiacus.**

3. **Gastrodiscus secundus.**

These parasites are about half an inch long, somewhat resembling an oval disc with convex dorsal and flattened or slightly concave ventral surfaces. They are rose red in colour. They possess numerous small papillæ on the ventral aspect. These parasites have been met with in Egypt, Senegal, Guadaloupe, and India, and infest the small intestines of horses, asses, and mules. They are occasionally found in the pharynx and in the rectum.

Symptoms.—These are not always sufficient to permit of diagnosis during life. If a number of parasites are present they may set up anæmia and marasmus; and in several instances of sudden death these parasites have also been found, and rightly or wrongly have been regarded as the cause of death.

Treatment is the same as for amphistomes and flukes, namely, the internal administration of filix mas followed by a physic ball and attention to possibly infested water-supply and pastures.

Distomum hepaticum (**Fasciola hepatica**).—This is the common so-called liver fluke, which may be up to an inch in length and half an inch broad. It is more commonly met with in sheep and cattle, but it is sometimes met with in the liver and bile ducts of the horse. It is rarely present in sufficient numbers to cause symptoms. It, however, may be occasionally responsible for jaundice. Its presence can only be definitely established by the discovery of eggs in the fæces. When present or suspected, treatment should consist of the administration of filix mas in repeated doses.

Parasites of the Intestines of Ruminants

As already mentioned in connection with parasites in the stomach a number of strongyles may be met with in the upper part of the small intestine of ruminants. Most of these have their normal habitat in the abomasum, but are carried into the intestines and there they continue their existence. Of that list, however, *Cooperia oncophora* (*Strongylus ventricosus*) and

Nematodirus filicollis (*Str. filicollis*) are found only in the intestines, but as they rarely occur alone any symptoms which are set up may be due to the combination of gastric strongyles with the others (see p. 314).

Ascaris vitulorum.—This worm is reddish white in colour and may be from 7 to 12 inches long. It is most frequently met with in calves in the small intestines and occasionally in the fourth stomach. It rarely affects adults, and is met with generally in the South of France.

Symptoms.—When very numerous it may give rise to loss of condition and occasionally to colic.

Treatment.—This consists in the administration of simple vermicides as in the case of ascaris in the horse, namely, linseed oil and turpentine or lysol to be followed by a saline purgative.

Ascaris ovis.—This is an ascaris which has once been met with in the sheep. It measures 5 to 6 inches in length, and is possibly *Ascaris vitulorum*. No symptoms have been recorded.

Trichocephalus affinis (*Trichuris ovis*).—This is a small worm which may be up to an inch in length and is met with in sheep and cattle with very wide distribution. Its chief habitat is the cæcum, and it is often in company with the tapeworm (*Tænia expansa*, *Anoplocephala expansa*, *Moniezia expansa*). *T. affinis* is the so-called " whip-worm," the anterior part of the body being attenuated and the posterior part thick. It is found with its head buried in the mucous membrane.

Symptoms.—Unless present in very large numbers this parasite apparently causes no symptoms. If very numerous, they may set up loss of condition, wasting, diarrhœa, and anæmia. These symptoms, however, are very likely to be caused by strongyles in the stomach, which are so often present simultaneously with whip-worms in the cæcum.

Treatment.—When symptoms have been observed the same treatment should be adopted as recommended for parasitic gastritis. The best results will be obtained by the combination of arsenic, copper sulphate, and iron. The animal should be taken up from the pastures if possible, and should be given a generous diet of trough foods.

Œsophagostomum columbianum.—This parasite is the cause of so-called *pimply gut* in sheep and cattle. In the latter animals it has been called *Œsophagostomum radiatum*. It was first described as occurring in Columbia, U.S.A., but is now also met with in Great Britain. It is probable that infection was introduced by means of manure from American cattle ships. The adult worm is from half an inch to three-quarters of an inch long, and is met with free in the large intestines of sheep and in the small intestines of cattle. The embryos, which are about ⅙th to ⅛th of an inch long, are met with in the substance of the bowel wall in the large and small intestines, where they form small nodules varying in size up to half a hazel nut.

Symptoms.—Unless these worms are present in considerable numbers they apparently do not give rise to any appreciable symptoms. On the other hand, when very numerous, they give rise in the late summer and early autumn to gradual and progressive wasting with irregular bowels and anæmia. The symptoms, however, are not sufficient to afford the basis for definite diagnosis. They are most frequently met with in the slaughter-house when the intestines are being prepared for sausage skins, for which purpose they are rendered useless.

Treatment.—There is no satisfactory treatment for the condition since the chief trouble is caused by the presence of embryos in the substance of the bowel. At the present time there are no remedies which can be brought into contact with them in this situation.

The life-history of the parasite is not well understood, but it is probable that a portion of its existence is passed in the pastures from which infestation takes place. Preventive measures may therefore be adopted with regard to the pasture on similar lines as recommended for gastric strongyles.

Sclerostomum hypostomum.—This is a parasite sometimes met with in the large intestines of the sheep and goat. It is up to one inch in length. It appears to pass part of its life-history in the substance of the bowel wall, where it may be seen in small nodules with minute punctures. It causes a hæmorrhagic enteritis with chocolate-coloured exudate, in which the parasite may be found.

Symptoms produced are those of progressive and intense anæmia, with emaciation, pallid and watery mucous membranes, irregular bowels, and debility.

Treatment for the condition is inadequate, but arsenic, antimony, and copper sulphate may be tried.

Cestodes or Tapeworms in Cattle and Sheep.—A number of tapeworms may be met with in cattle, sheep, and goats. In the former there are four or five, in sheep twelve or thirteen, and in goats two. They are all unarmed, and rarely give rise to any serious symptoms. The chief ones consist of *Moniezia expansa* or *Anoplocephala expansa*:

Mon. or *Anop. alba*
Mon. or *Anop. denticulata* } All met with in both the ox and sheep.
Mon. or *Anop. planissima*
Thysanosomum actinioides (*Tænia fimbriata*), met with in sheep.

Of these, the *M. expansa* is the longest, and may be up to 20 feet long. It is probably the longest known tapeworm. The others may attain the length of about 6 ft. (for full list of these tapeworms see p. 538).

Symptoms.—In cattle they rarely set up symptoms, but they have been known to cause colic and complete obstruction of the bowels. In sheep they are more likely to cause injurious effects, particularly when present in large numbers, and especially affecting lambs. The symptoms are set up in the late autumn and throughout the winter. The animals are unthrifty, fail to fatten, their fleece is depreciated in quality and quantity, and chronic diarrhœa may supervene. Death may result from anæmia. In such cases the tapeworms are generally accompanied by one or more varieties of round worms. Positive diagnosis may be made by discovery of the segments in the fæces or by means of post-mortem examination.

Treatment of Tapeworm in Ruminants.—These worms are comparatively easily removed since they are unarmed and are attached only by suckers. The affected animal should be put on a course of areca nut, which must be freshly ground. For cattle ʒiii. or ʒiv. and for sheep ʒi.-ii. should be given twice daily for a week and then followed by a simple purgative of sulphate of magnesia or common salt, or linseed oil and turpentine. Other agents which have been recommended in addition to areca nut for sheep are:

Kamala ʒi.-iss. twice in the same day.
Arsenic gr. v.-vii. once daily for a week.
Filix mas ʒi. daily for a week.
Picrate of potash gr. x.-xx. daily for a week.
Naphthalin gr. xv.-xx. daily for a week.

Each of these should be followed by a purgative at the end of the course. Affected sheep should be taken up from the pasture while under treatment, and since the life-history of the parasites is unknown, pastures known to be infested should as a precaution be dressed either with lime or salt, or if possible put under the plough.

Trematodes in the Intestines of Ruminants.—The normal habitat for most of these parasites in ruminants is the liver, but occasionally one or other may be met with in the small intestines, apparently having passed down the bile ducts. In this situation they do not give rise to any particular trouble, the ill-effects due to flukes being entirely due to infestation of the liver.

Intestinal Parasites in Swine

Nematodes or Round Worms.—The pig harbours a number of round worms in the intestines of some importance. The chief ones are:

Ascaris suis.
Echinorhynchus gigas.
Trichocephalus crenatus.
Trichinella spiralis.

Ascaris suis.—This parasite occupies the small intestines of pigs and is sometimes present in very large numbers. It may attain the length of 8 to 10 inches with the thickness of a goose quill. It is most serious in its effects on young pigs, and may actually cause a complete obstruction in the small intestines. Specimens have occasionally been found in the bile ducts.

Symptoms.—If these worms are present in small numbers there may be no ill-effects. When in larger numbers they give rise to unthriftiness, loss of condition, and weakness of the hind limbs. Frequently brain symptoms are set up in the form of convulsions or fits, in which the animal falls to the ground and struggles, recovering in a short time. Tenesmus may also be set up, resulting in intussusception and eversion of the bowel. In other cases the parasites give rise to a suddenly occurring obstruction of the bowels with acute colic and may prove fatal.

Treatment.—The worms may be got rid of by the administration of calomel gr. ii. or iii. up to gr. x. with jalap ʒi.-ii., which should be mixed with treacle and placed in the mouth with a flat stick, or castor oil and turpentine may be used. Oil of chenopodium ♏i. per lb. body-weight given fasting is very serviceable. Where brain symptoms are exhibited bromide of potassium should be given in doses from ʒss. to ʒii. according to the age and size of the pig. If pigs are in good condition, it may be well to consider the advisability of slaughter.

Echinorhynchus gigas.—This is a big cylindroid worm, frequently annulated, and may be from 5 or 6 inches (males) up to 15 or 18 inches long (females). It is a dirty white or slightly greenish colour, and is met with chiefly in the small intestines, especially in the duodenum. It may be free in the bowel or more often attached by means of its hooks to the mucous membrane. Sometimes they may actually pierce the mucous membrane and produce a small abscess which may result in perforation and set up peritonitis. The parasite has a somewhat complicated life-history and passes some of its stages in the so-called maybug (or cockchafer).

Symptoms.—Young pigs are much more seriously affected than adults, and some years these parasites are responsible for considerable mortality. If the worms are not numerous there may be no symptoms shown, but if they are present in considerable numbers they give rise to loss of appetite, abdominal pain, signs of colic, general wasting, and sometimes colic. In some instances brain symptoms are exhibited, such as epileptiform fits and convulsions, which may terminate fatally. Definite diagnosis may be made by the microscopic examination of fæces for eggs which have a characteristic appearance. The injury to the intestinal wall ruins it for sausage skins. (The condition has sometimes been called *pimply gut* by butchers, but should not be confused with œsophagostomum in sheep and cattle.)

Treatment.—It is not easy to definitely expel these worms. Such agents may be used as calomel and jalap, or santonin, or oil of turpentine as in the case of *Ascaris suis*. Attention should also be paid to the destruction of fæces containing eggs either by burning or burying with lime.

Trichocephalus crenatus (*Trichuris suis*).—This worm closely resembles the *T. affinis* of cattle and sheep, and is found principally in the cæcum. It rarely causes any symptoms, and its presence is rarely suspected until its discovery in the abattoir, and therefore does not call for treatment.

Trichinella spiralis (*Trichina spiralis*).—This is a very small nematode worm, the adult stage of which is about ⅛th of an inch long, and is met with in the intestines of many mammals, but particularly the pig. In this situation it may cause symptoms, but its chief importance attaches to the fact that its larvæ or embryos migrate into the muscular tissues, where they become encysted, and in this situation are liable to infect human beings eating such pork in a raw or imperfectly-cooked state.

Symptoms.—When present in considerable numbers in the bowel the trichinæ may set up irritation with general depression and fever, indisposition to move about, and sometimes diarrhœa. This condition lasts for several weeks and is followed by muscular stiffness which marks the invasion of the tissues with the larvæ. Pigs then become seriously ill; they appear to be paralysed, and movement causes considerable pain. There may be difficulty in swallowing.

Diagnosis during life is almost impossible, and therefore no *curative treatment* can be adopted.

Prevention may be practised by destroying all rats which may be intermediate carriers of the parasite, and by boiling all butchers' offals fed to swine. This condition assumes greater importance from the point of view of meat inspection, and is considered in greater detail when dealing with parasites of the muscular tissue.

(A complete list of nematodes of the pig will be seen on p. 541.)

Cestodes.—Except for a record of *Thysanosoma giardi*, normally a parasite of cattle, there are no tapeworms met with in the intestines of swine.

Trematodes of the Intestines of Swine.—Two trematodes are frequently met with in the intestines of pigs in the Far East.

Fasciolopsis buski.—This fleshy parasite, from 1 to 2 inches long by ½ inch broad, is commonly met with in large numbers in the duodenum of pigs in South China.

Gastrodiscoides hominis.—This occurs in the cæcum of 5 per cent of the pigs in French China. They closely resemble the Gastrodiscus found in the horse, but do not possess papillæ on their ventral aspect, and are about one-third their size.

Intestinal Parasites of the Dog

Nematodes or Round Worms (see p. 542).—The intestines of the dog harbour a number of round worms which, as in the case of the larger animals, frequently give rise to no clinical symptoms by which their presence can be diagnosed. Two of them, however, may set up serious clinical conditions, and call for special attention.

Ascaridiasis.—Two genera of Ascaris are met with in the dog, namely:

(*a*) *Belascaris* (*Ascaris marginata*, *Belascaris marginata*).
(*b*) *Toxascaris* (*Toxascaris limbata*).

These are fairly long round worms, the former being white and up to four or five inches in length, the latter pinkish white, two to three inches long. They are both common parasites, and are met with in dogs of all ages, but give rise to symptoms most frequently in puppies. Their normal habitat is the small intestines.

Symptoms.—The first indications are distended abdomen (pot-belly) and irregular bowels with intermittent diarrhœa. Occasionally worms are passed with the fæces. There may be general unthriftiness and emaciation in spite of a good appetite and a generous supply of good food. The coat is frequently dry and harsh, and there is often some skin irritation. In many cases the worms pass forward into the stomach, causing irritation and vomiting of parasites. Nervous complications very frequently arise and may take the form of frenzy with fits of screaming or yelling for which no reason is apparent. At other times there are convulsions resembling epileptiform seizures which sometimes terminate fatally. In rare cases the parasites may be so numerous as to cause impaction of the bowel, and in other cases they may set up intussusception and tenesmus with eversion of a portion of the bowel. Microscopic examination of the fæces reveals the presence of eggs, which will confirm the diagnosis if parasites have not actually been seen. Symptoms may be met with in very young puppies within two or three weeks old, infection having probably taken place by the ingestion of eggs which may have been passed by the mother and have contaminated the teats or bedding.

Treatment.—In adults a variety of vermicides may be used, such as santonin, filix mas, oil of chenopodium, areca nut, pelletierin, and turpentine. Oil of chenopodium in doses of one minim per lb. body-weight may be given, and is the most effective agent.

Very good results are also obtained by the use of santonin, the dose of which may be calculated at one-tenth of a grain per lb. body-weight, regarding three or four grains as the maximum dose for a large dog. It should be given in combination with a purgative such as calomel or should be followed by a purgative. In very young puppies, or in delicate toy dogs, the safest treatment consists of a course of syrupus ferri iodid. mm. x.-xv. to be administered twice daily on an empty stomach in emuls. petrol. ʒi.-ii. Another useful combination for young puppies consists of ol. chenopod. mm. ss., ol. tereb. mm. ii., ol. anisi mm. ii. with ol. ricini ʒss. It is important that whichever vermicide is used it must be administered on an empty stomach, the animal having fasted twenty-four hours, and should be followed by a purgative. Nervous symptoms should be controlled by the administration of potassium bromide or syrup of chloral.

Ankylostomiasis.—This condition, sometimes known as hook-worm disease, is set up by four parasites, namely:

Ancylostoma trigonocephalum (*Ancy. caninum*).
Ancy. ceylonicum.
Ancy. brasiliense.
Uncinaria stenocephala.

The parasites are small white or reddish white worms from a quarter to three-quarters of an inch long. The adult worms are met with in the small intestine of dogs, where they bite rather deeply into the mucous membrane and so become blood-suckers. The earlier phases of development take place outside the body, and the later larval stages are met with in the lungs and in the blood, and in the gastric mucous membrane. The chief method of infection is probably through the skin, but they may also be taken in by the mouth in infected water.

Symptoms.—These parasites set up great emaciation, debility, and weakness, which becomes progressive and develops into a pernicious anæmia. The appetite is well maintained, but the dog becomes listless and easily fatigued. There is frequently irritation of the skin, which becomes reddened and sometimes œdematous and ulcerated. There is a purulent discharge from the nose with periodical attacks of bleeding (epistaxis). The bowels are irregular, with a tendency to diarrhœa and dysentery. The anæmia becomes very intense, with pallid mucous membranes, and a great reduction in the red blood corpuscles, which may become reduced to one-half or one-third their normal number. The condition may last for months, and ultimately terminates in coma and death.

Diagnosis can be confirmed by the microscopic examination of the fæces for eggs. The disease sometimes occurs as an enzoötic, attacking packs of hounds.

Treatment.—Isolate or segregate affected animals and destroy their fæces by burning. Provide them with a clean water-supply, and practise thorough disinfection of the premises to destroy eggs or embryos. Stagnant water should be drained away or filled up, and infested zones dressed with salt or lime. Medicinally, vermicides should be given internally, such as kamala up to ʒi. with gr. ii.-iii. of calomel. Filix mas, which has been found serviceable in man, is also effective in the dog. Arsenic, thymol, and beta naphthol also give good results. A generous diet must be given, followed by a course of tonics, of which syrup. ferri phos. co. with cod-liver oil and malt is about the best.

Another parasite met with but rarely causing symptoms is *Trichocephalus depressiusculus* (*Trichuris depressiusculus*) in the cæcum.

Cestodes or Tapeworms of the Dog (p. 541-2).—The dog harbours a large number of tapeworms, all of which are met with in the small intestines. Occasionally they are found in the stomach.

Tapeworms have a complicated life-history, needing two hosts for their complete development. The *Tænia serrata* affords a good example, and in a general way the others conform to the same type. The adult *Tænia serrata* consists of head and segments in the intestine of the dog. Segments become fertilized, develop considerable numbers of eggs, and when mature they become detached and are passed out of the intestines and for a time possess spontaneous wormlike mobility. The eggs are liberated by rupture of the segment, and contaminate either food or water which is ingested by the second or *intermediate host*—a rabbit or hare in the case of the *T. serrata.* In the stomach, embryos are hatched out from the eggs and migrate to various parts of the body to their predilection seats—to the mesentery in the case of the worm in question. Here they become arrested and each embryo develops into a "cyst," "hydatid" or "bladder-worm" called the *Cysticercus pisiformis.* This is the *intermediate stage* of the parasite, and if it is eaten by a dog it passes into the intestines and develops into a mature *T. serrata.* The dog is thus the *ultimate* or *definitive host.*

The principal tapeworms of the dog and cat, together with their intermediate stages when known, can readily be seen in the following table:—

Adult Tapeworm.	Length.	Armed or Unarmed.	Host.	Intermediate Stage.	Habitat of Intermediate Stage.	Intermediate Host.
Tænia serrata . .	2 to 6 ft.	Armed	Dog	Cysticercus pisiformis	Peritoneum	Rabbits and hares.
„ marginata .	4 „ 6 ft.	„	Dog	Cyst. tenuicollis	Peritoneum and liver	Ruminants and pigs.
„ echinococcus .	up to ¼ inch.	„	Dog and cat	Echinococcus veterinorum	Wide dissemination, chiefly liver and lungs	Most mammals (including man).
„ ovis . . .	2 to 3 ft.	„	Dog	Cyst. ovis	Muscle	Sheep.
„ crassicollis .	6 in. to 2 ft.	„	Cat	Cyst. fasciolaris	Liver	Rats and mice.
„ cœnurus . . (Multiceps multiceps)	1 to 1½ ft.	„	Dog	Cœnurus cerebralis	Cranium	Sheep and other ruminants.
„ serialis (M. serialis)	1 „ 2 ft.	„	„	Cœnurus serialis	Intermuscular and subcutaneous connective tissue	Rabbits and hares.
Dipylidium caninum (T. cucumerina) T. elliptica	up to 1½ ft.	„	Dog and cat	Cryptocystis trichodectis	..	Louse and flea of dog.
Mesocestoides lineatus (T. litterata) . .	2 to 6 ft.	Unarmed	Dog and cat	..	..	..
Dibothriocephalus latus . . .	20 „ 30 ft.	„ „	Dog and cat	Plerocercoid	Internal organs	Various fish.
Diboth. cordatus . .		„	Dog	..	..	..
„ fuscus . .		„	Dog	..	..	..

In the case of the *D. latus* it has been recently proved that two intermediate hosts are required, first a cyclops and second a fresh-water fish.

Symptoms.—Dogs very frequently harbour these various tapeworms in small numbers without giving rise to any symptoms beyond the passage of an occasional segment with the fæces. Sometimes segments may be seen in the animal's bed, partially dried, almost resembling small barley grains. When present in large numbers they may cause variable appetite, loss of condition, irritability of the skin with scratching, irregular bowels, and pruritus ani. The anal glands may also be impacted, and on squeezing them out segments of tapeworm may be expelled. In the case of *Dibothriocephalus* instead of individual segments a shred consisting of several empty segments may be seen. In cases where the tapeworms are abnormally present in the stomach, vomition may occur. Convulsions are occasionally set up and excitement sometimes amounts to frenzy, but much less frequently than in ascaridiasis.

Treatment.—First fast the patient for fifteen or twenty hours and administer a purgative to clear out the alimentary tract. When this has been effected a vermifuge should be administered. The best results are obtained by freshly grated areca nut, gr. ii. per lb. body-weight, with ʒii. maximum dose for a large dog, or tenaline (an ethereal extract of areca-nut) mm. iss. per lb. body-weight, regarding mm. xv. as the smallest dose and ʒiss. as the maximum dose. The tenaline should be made into a draught with glycerin ʒss. and water ʒii. The dog should be led about to prevent vomition, and if he has been properly prepared, the dose should be effective in from fifteen to thirty minutes. If it is not effective an enema should be given. This frequently causes the expulsion of considerable numbers of the tapeworms. Other useful preparations are extract of filix mas mm. xx. to ʒi., ol. tereb. ʒi.-ii. in emulsion, tannate of pelletierin gr. v., ol. chenopod. mm. vi., and kamala ʒss.-i. These tæniafuges should be repeated in a week or ten days. Some dogs persistently vomit the tæniafuge. This can be controlled either by administering a preliminary dose of cocaine gr. ss.-i. or chloretone to anæsthetize the stomach, or by giving a hypodermic injection of morphia half an hour before the worm medicine. If convulsions are present bromides should be given. Frenzy is best controlled by morphia. In cases where debility has been produced, a course of tonics should be given, such as syrup. ferri phos. co.

Preventive measures can only be adopted by taking into account the life-histories of the tapeworms where known, and consist solely of avoiding in a raw state those foods which harbour or contain the hydatid or bladder-worm stage. This would involve the boiling of all butchers' offals or fish fed to dogs. It is doubtful, however, if it is worth while undertaking this procedure, since the ill-effects caused by the tapeworms themselves are rarely severe. Greater importance, however, attaches to the case of the *Tænia echinococcus* on account of the possible serious ill-effects in man resulting from ingestion of any foods contaminated with ova of this parasite, since the human being may become the abnormal host of the intermediate stage of this tapeworm. Moreover, owners of dogs infested with *Tænia echinococcus* may contaminate their hands, etc., when caressing their dogs or allowing themselves to be licked by dogs, and in this way and also by the objectionable habit of kissing dogs, infect themselves.

In the case of *Dipy. caninum* preventive measures entail the freeing of dogs of external vermin such as lice and fleas—intermediate hosts of the parasite.

Trematodes in the Intestines of Dogs.—Trematodes are not very common in dogs except in Egypt and the Far East, particularly Japan, China, India. They include:

Heterophyes heterophyes.
Echinostoma perfoliatum.
E. trigonocephalum.
Hemistoma alatum.
Yokogawa yokogawai.
Ascocotyle minuta.
(For complete list, see p. 541.)

They are of no special clinical importance, as they appear to cause no symptoms when present in the intestines, and are therefore not diagnosed during life. They are all very small, and may even escape observation on post-mortem examination. If their presence is suspected, treatment should consist of repeated doses of castor oil and extract of filix mas. They are probably all acquired from ingesting fresh-water fish infected with encysted larvæ.

Intestinal Parasites of the Cat

Nematodes.—There are a few round worms infesting the cat, but only three of practical importance, namely:

Ascaris mystax (*Belascaris cati*).
Ancylostoma trigonocephalum (*A. caninum*).
Ollulanus tricuspis.

The following nematodes may be met with, but rarely or never produce any clinical symptoms:

Chlamydonema felinium.
Oxyuris compar.
Trichosomum lineare (*Capillaria lineare*).
Trichocephalus serratus (*Trichuris serratus*).
T. campanula.
Trichinella spiralis.

Ascaridiasis in the Cat.—This is due to the *Ascaris mystax* (*Belascaris cati*), which is a thin round worm from three to five inches long, and like the ascaris of the dog is frequently curled round. It usually infests the small intestines, but is occasionally met with in the stomach.

Symptoms set up correspond very closely with those of ascaridiasis in the dog and consist mainly of loss of condition and distended abdomen with irregular bowels and occasional vomiting. Fæces and vomit may contain parasites. Eversion of the bowel and intussusception are sometimes produced. Brain symptoms are rarely set up in ascaridiasis of the cat.

Treatment.—The same remedies and doses may be used for the cat as for dogs of the smaller breeds, but for young kittens half the doses should be employed.

Ankylostomiasis.—This condition consists of infestation with *Ancylostoma caninum*, and appears to be less commonly met with in the cat than in the dog. It gives rise to similar symptoms of debility and progressive anæmia with irregular bowels, skin irritation, and nasal discharge, which may be blood-stained. Vomition also frequently occurs, both the vomit and the diarrhœa being dark in colour, almost black.

Treatment is the same as for ankylostomiasis in the dog, the best remedy being castor oil and liquid extract of filix mas.

Ollulaniasis.—This is a very rare condition set up by *Ollulanus tricuspis* in the cat. The adult worm is very small, rarely exceeding $\frac{1}{25}$th inch long. It is met with in the stomach and intestines, and may set up gastritis. A more serious phase arises during the migration stages of the larvæ when passing through other tissues of the body such as lungs, diaphragm, and muscles where they ultimately become encysted. During migration they cause fever and intense muscular pains which may give rise to a diagnosis of acute rheumatism.

Treatment.—No curative measures can be adopted, but as the condition is never diagnosed during life they are not likely to be called for. Symptoms of gastritis should be treated with gastric sedatives as bismuth carbonate or chloretone; and the painful condition of the muscles may be relieved by analgesics such as aceto-salicylic acid. Preventive measures may be adopted, and consist in the extermination of rats and mice which are also hosts of the worm, and from which cats usually become affected.

Cestodes or Tapeworms of the Cat.—The cat is the host of two common tapeworms, namely:

Tænia crassicollis.
Dipylidium caninum (*D. felis. Tænia elliptica*), which also affects the dog.

The following parasites of the dog are occasionally found also in the cat:

Tænia echinococcus.
Mesocestoides lineatus (*Tænia litterata*).
Dibothriocephalus latus.

Tænia crassicollis, which is peculiar to the cat, is a rather thick tapeworm measuring from six inches to two feet long. Its cystic stage is called the *Cysticercus fasciolaris* and is found in the liver of rats and mice.

Symptoms of Tapeworms in the Cat.—Unless tapeworms are present in considerable numbers they rarely give any trouble. The worst effects are produced by the *T. crassicollis*, and are more likely to be met with in kittens than in adults. They may cause great loss of condition, and irregular bowels, and diarrhœa, and sometimes vomiting. In rare cases tapeworms have actually been included in the vomit, but these parasites are much less frequently vomited than round worms. When in very large numbers they have been known to cause death.

Treatment.—The same remedies may be used as in the dog in relatively smaller doses, the best results being obtained by areca nut or tenaline.

Trematodes of the Intestines of the Cat.—Intestinal trematodes are rare in the cat, and never set up clinical symptoms. They include

Heterophyes heterophyes } both in Egypt.
H. æqualis }
Loosia parva in the Balkans.

It is probable that as in the case of the dog they are acquired from infected fresh-water fish.

G. H. W.

DISEASES OF THE LIVER

Congestion of the Liver

This may be referred to under three headings according to the system of vessels affected. There may be

(*a*) **Active congestion,** when an increased amount of blood is brought to the liver by the hepatic artery.

(*b*) **Passive congestion,** when there is interference with the circulation of the portal vein and its branches.

(*c*) **Biliary congestion,** when there is interference with the outflow of bile from the liver.

Active Congestion of the Liver ; Arterial Hyperæmia

This condition is very rarely diagnosed in the domesticated animals. It may be brought about by irritating and stimulating materials absorbed from the alimentary tract and especially excessively nitrogenous foods and brewers' and distillers' grains. It is also conduced to by lack of sufficient exercise, and is consequently most frequently met with in animals that are being overfed and under-worked and prepared for show purposes. A similar condition may be regarded as a normal physiological one after meals when the liver is called upon to exert its normal functions. When this condition is prolonged then it becomes pathological. Active congestion may be preliminary to the development of acute hepatitis.

The *symptoms* set up are very indefinite, and may consist of some slight abdominal pain or uneasiness, which is most evident after meals with a slight rise of temperature and general depression. This is followed by the general symptoms of indigestion. It rarely, however, sets up symptoms by which it can be definitely recognized.

Treatment consists in the administration of a mild saline purgative and restriction of the diet, which should be of a laxative character.

Passive Congestion of the Liver ; Chronic Venous Congestion ; Portal Congestion

Passive congestion of the portal vein and its branches may be caused by anything which diminishes the flow of blood from the liver on the one hand, or anything which diminishes the force of the blood travelling up the portal vein to the liver. Examples of the former are met with in incompetence of the right side of the heart causing regurgitation into the veins ; chronic obstructive lung disease ; pressure on the posterior vena cava or on the hepatic vein by morbid growths of any kind, such as tumours or enlarged glands. The rate of flow of portal blood to the liver may be reduced by debility of the left side of the heart, or congestion of vessels in the abdominal area from which the portal blood is supplied. Portal congestion is met with in animals with a so-called lethargic temperament, and thus is common in animals having insufficient exercise, as is often the case in old dogs. The portal circulation may also be slowed down as the result of absorption of deleterious substances from the alimentary tract.

Symptoms.—These are ushered in by general dulness and depression followed by signs of indigestion ; irregular appetite and bowels with a tendency to constipation, the fæces being offensive in odour due to the insufficient secretion of bile—the natural disinfectant of the bowels. The mucous membranes are dirty yellowish in colour, and the pulse is soft and slow, lacking tone. The head is often hung down and the eyelids drooping. Vomiting in dogs and cats is frequently met with. There is frequently a slight rise of temperature, but it is never excessive. The urine is of a higher colour than normal. There is usually some enlargement of the liver, which may be felt on manipulation of the abdominal wall on the right side immediately behind the ribs in the large animals and producing dulness on percussion. In the smaller animals, the dog and cat, the enlarged liver can be more easily felt on manipulation of the abdomen. Slight congestion of the liver is a frequent complication of fevers in most animals. On further examination one may find the cause of the congestion and the symptoms dependent thereon, such as jugular pulse with valvular disease of the right side of the heart, œdema of extremities, and dropsy of the peritoneum. The latter condition is comparatively common in the smaller animals, but not so frequently met with in the larger animals.

Treatment : Horse.—First give a purgative. Calomel probably gives the greatest relief. ʒi.-ii. administered in a mash, or calomel ʒi. with aloes ʒiii. in bolus. This may be followed on subsequent days with magnesium sulphate ʒiii.-iv., and may be combined with ammonia and nux vomica.

For cattle and sheep a combination of magnesium sulphate and sodium sulphate gives the best results, followed by ammonium carbonate and nux vomica.

For dogs and cats it is a good plan first to give an emetic such as apomorphine hydrochlor. gr. $\frac{1}{16}$ to $\frac{1}{8}$ hypodermically. The act of vomition exerts mechanical pressure on the liver and may thus reduce the congestion and accelerate the circulation. This should be followed by calomel gr. i.-iii.; subsequently magnesium sulphate or sodium sulphate may be given daily in small doses. Hydrarg. cum creta is often useful. If the heart is affected digitalis should be given. When dropsy is present the abdomen should be tapped, the water-supply curtailed, and a course of potassium iodide, potassium nitrate, and digitalis administered. General depression is frequently relieved by the combination of ammonium carbonate or aromatic spirits with the other remedies prescribed.

Attention must be paid to the diet, which should be somewhat restricted for a day or two and of a laxative easily digested character.

Biliary Congestion ; Jaundice ; Icterus

This condition may be brought about by obstruction of the bile flow or by the reabsorption of bile after it has been secreted. The cause may consist of catarrh of the bile ducts, which is frequently secondary to muco-enteritis; parasites in the bile ducts, such as flukes and round worms; gallstones and inspissated bile. The ducts may also be occluded by cysts or tumours in the liver tissue, which cause pressure on the outside of the duct. Cirrhosis, tuberculosis, bacterial necrosis, fatty degeneration, and amyloid degeneration may set up subacute jaundice. Acute jaundice may also be set up in the course of certain specific affections such as piroplasmosis, influenza, and distemper, and also in azoturia in horses. The yellow pigmentation met with in piroplasmosis and azoturia is largely due to the presence in the blood plasma of free blood pigments liberated by the destruction of red blood corpuscles in the former and of red muscle fibres in the latter.

Symptoms.—These depend upon absorption of bile and the arrest of the bile functions in the bowel. Symptoms may arise in some instances rapidly and acutely, in other cases the onset is subacute. Acute jaundice frequently occurs in the horse during influenza and in the dog during distemper. The most pronounced feature is the sudden yellow discoloration of the skin and mucous membranes. It may be observed first in the conjunctiva and sclerotic. There is often an acute rise of temperature, which may be up to 106° F. or 107° F., but this is not so pronounced in subacute or chronic forms of jaundice. There is very great dulness and depression, and rapidly progressive debility and emaciation. The urine is rich yellow or yellowish green and contains bile salts and bile pigments. Constipation supervenes owing to the absence of bile from the bowels, and the fæces are offensive in odour and clay-coloured. In carnivores acute jaundice frequently induces fits or convulsions and repeated vomiting, the vomit being either yellow or greenish in colour, and if not speedily relieved may prove fatal. If the condition lasts long anæmia is set up with œdema of the abdomen and pendulous parts. If the œdematous liquid is removed it is also bright yellow in colour, containing bile pigments. Bile pigments can be demonstrated by Gmelin's test or by

Rosenbach's Test for Bile Pigments.—This consists of passing a little of the suspected urine through filter paper. A portion of the paper is then placed on a white porcelain dish and is touched by a glass rod which has been dipped in fuming nitric acid. If bile pigments are present a ring of colours is produced at the spot, beginning with yellow and passing on to violet, brown, and green from within outwards. In some cases only the green colour is produced.

Treatment of Jaundice.—If the condition is due to biliary congestion first give a purgative, the best for the purpose being calomel. This may be preceded by an emetic such as apomorphine in the case of animals that can vomit. The act of vomition thus produced may dislodge the obstruction in the bile duct and permit of the resumption of the flow of the bile. Following the purgative a biliary depressant may be given, such as opium in small doses or the combination of small doses of calomel and opium. In the case of dogs, pulv. ipecac. co. gr. v.-x. twice or three times daily is very useful. Repeated small doses of magnesium sulphate are often very serviceable, and the injection of normal saline solution into the subcutaneous tissues or even per rectum often affords good results. In the smaller animals where collapse is threatened stimulants should be given, such as ammonia and nux vomica or brandy; or liq. strychninæ may be given hypodermically. When jaundice is due to other causes, such as piroplasmosis or azoturia, corresponding treatment must be adopted, and the reader is referred to the chapters on those subjects. Diet must be of easily digestible character—in horses and cattle limited quantities of the best hay and boiled linseed or soaked linseed cake, and a little green food if available. For dogs, boiled sheep's paunches are to be recommended, with barley water and lime water or normal saline to drink.

Gallstones ; Biliary Calculi ; Cholelithiasis

Gallstones are concretions or incrustations which make their appearance in the bile ducts or sometimes in the gall-bladder. They are met with most frequently in animals possessing a gall-bladder, and more rarely in the horse. They usually consist of bile salts deposited round a small nucleus consisting of bacteria or a little epithelium detached as the result of catarrh of the bile duct. Occasionally the starting-point may be a parasite. Anything which causes obstruction of the flow of bile tends to the formation of calculi. Gallstones vary considerably in size and may be very small like shot, in which case they are usually multiple and may actually run into hundreds. In other cases they are as thick as one's finger, forming casts of the larger bile ducts, and may on occasion attain the size of one's fist. They are usually reddish brown in colour, but sometimes they are greenish or yellowish brown, and at other times almost white like chalk. They are not very dense, and when dried will float on water. The outer surface may be smooth or slightly bosselated. When cut across they are seen to be laminated as though made up of superimposed layers. In cattle they are frequently large and soft and consist principally of inspissated bile. The chemical composition is not constant, but consists principally of cholesterin with glycocholates and taurocholates of soda, carbonate and phosphate of lime, together with some mucus and fat.

Symptoms.—It is frequently the case that gallstones are found in the gall-bladder of animals on post-mortem examination without their having been suspected during life, and occasionally they have also been found in the bile ducts in horses without having given rise to any symptoms. In the majority of cases, however, they cause obstruction to the flow of bile, and set up biliary congestion and jaundice, which is sometimes acute and at other times subacute. The symptoms are then the same as described in the previous chapter. On other occasions when the calculi are passing down the bile ducts they may set up intense pain and colic, and the seizures may be intermittent and recurrent. This has been described as *hepatic colic.* In rare cases the gall-bladder has been known to rupture, setting up intense jaundice, collapse, and death in one or two days.

Treatment.—Owing to the difficulty of definitely diagnosing gallstone, the same treatment should be adopted as recommended for biliary congestion, relying principally upon the combination of calomel and opium. In cases of hepatic colic the pain may be controlled by the hypodermic injections of morphia or by the administration of chloral hydrate. It is said that repeated doses of olive oil assist the passage of gallstones. In dogs, if gallstone is diagnosed, surgical removal may be resorted to.

Hepatitis

Three forms of hepatitis may be recognized, namely:

Acute parenchymatous hepatitis.
Suppurative hepatitis.
Chronic interstitial hepatitis or cirrhosis of the liver.

Acute Parenchymatous Hepatitis.—This condition is rarely met with, and when it does occur it is usually preceded by active congestion of the liver. It may be brought about by poisonous substances absorbed from the alimentary tract, such as bacterial toxins, poisonous foods, poisonous plants, phosphorus, and arsenic. It may also occur in the migration stages of certain parasites, such as *Distomum hepaticum*, *Cysticercus tenuicollis*, etc. It has also been stated to have been caused by plants of the genus *Senecio* in South Africa associated with catarrh of the stomach and intestines.

Symptoms.—These may not be sufficient to afford a positive diagnosis during life. There are usually loss of appetite and indisposition to move about, irregular bowels and yellowish mucous membranes; urine is high-coloured, and there is elevation of the temperature two or three degrees. On manipulating the abdomen pain is produced by pressure over the region of the liver towards the right side, particularly in the smaller animals. There is also some pain evidenced in defæcation. Lameness of the right fore limb is often said to be associated with acute affections of the liver. It may be present in some instances, but this symptom would appear to be assumed in many instances by analogy with the human subject, in whom pain under the right shoulder is a fairly constant symptom in acute liver affections. In poisoning by *Senecio* as occurring in South Africa there is also persistent diarrhœa with tenesmus, sometimes prolapse of the rectum and excitement.

Treatment.—First clear out the alimentary tract with a saline purgative. Follow this with small doses of opium as a hepatic depressant, the constipating effects of which should be controlled by a laxative diet and repeated small doses of salines. Where the active cause of the condition is discoverable steps must be taken to avoid further access to it.

Suppurative Hepatitis.—This condition is due to pyogenic organisms in the liver and is usually secondary to a suppurative condition in some other part of the body, such as abscesses, or strangles in the horse. It may also occur in navel affections in young animals and in parasitic affections of the liver. Pyogenic organisms may also reach the liver as the result of injuries by foreign bodies from the stomach, as nails, skewers, needles, etc. The lesions set up consist of abscesses of the liver.

Symptoms.—These depend upon the number and extent of the lesions. If the suppuration is limited in extent there may be no noticeable symptoms beyond some slight indisposition. When the abscesses are more numerous or develop rapidly, acute symptoms may be set up with abdominal pain, irregular appetite and bowels, fluctuating temperature, mucous membranes yellowish and in some cases distinctly jaundiced. When due to a foreign body the pain is more pronounced and there is indisposition to move, with pain in defæcation and in palpation of the abdominal wall in the region of the liver. In the case of the smaller animals vomiting may be set up. The condition may persist for several weeks, but usually terminates fatally either from rupture of the liver with internal hæmorrhage or from rupture of the abscess into the abdominal cavity setting up peritonitis.

Treatment.—This is not very satisfactory, and especially so owing to the difficulty of making a definite diagnosis. Surgical treatment has been recommended, but such treatment is not hopeful. If there is good ground for positive diagnosis slaughter is the best procedure.

Cirrhosis of the Liver.—This is a condition of *chronic interstitial hepatitis* in which there is marked increase of the connective tissue along the course of the bile ducts and between the lobules of the liver. As a general rule this results in marked enlargement of the liver (*hypertrophic cirrhosis*), but in other cases the liver becomes reduced in size owing to contraction of the connective tissue and destruction of liver cells (*atrophic cirrhosis*).

The commonest cause of this condition in the domesticated animals is the presence of parasites in the bile ducts and in the liver substance, such as distoma, echinococcus cysts, and tenuicollis cysts. Cirrhosis may also be produced by irritants absorbed from the alimentary tract, such as damaged foods, mouldy foods, or indigestible innutritious foods in low-lying marshy districts. Excessive feeding on brewers' and distillers' offals has been suggested as a cause. Certain plants may also set up cirrhosis of the liver, particularly those belonging to the *Senecio* group. In some cases, however, it is quite impossible to ascertain the cause. In the human subject alcohol is a common cause of cirrhosis, but this is unlikely to be the case in the domesticated animals.

Symptoms.—These are usually ushered in by general unthriftiness, which is slowly developed and may not be observed for some time. This is followed by signs of indigestion with loss of appetite and irregular bowels with offensive fæces, frequently mucus-coated. There are loss of flesh and pendulous abdomen. The mucous membranes are paler than normal and may be slightly yellow or in advanced cases markedly jaundiced, depending on the extent of the closure of the bile ducts. Animals become listless and depressed and are unable to undergo prolonged exertion. In hypertrophic cirrhosis the liver becomes much enlarged and may be felt on the right side in horses and cattle, immediately behind the last rib. In the smaller animals enlargement may be felt on both sides of the body, and also along the floor of the abdomen extending towards the umbilicus. Cirrhosis of the liver frequently gives rise to dropsy of the abdomen (ascites) and the general connective tissues, particularly of the limbs (anasarca). This complication occurs most frequently in dogs and cats, and generally runs a chronic course; in some cases, however, it runs a rapid course and produces collapse and death in a comparatively short time. The writer has seen it prove fatal after two days' illness in a cat with vomiting and collapse. In sheep affected with fluke disease progressive emaciation and dropsy are commonly produced, but in some instances no symptoms whatever are set up, and one is surprised in the abattoir to meet with badly affected livers in sheep in prime fat bodily condition.

Treatment.—Treatment of cirrhosis of the liver is only palliative. In the case of the larger animals it is rarely worth while to persist in treatment owing to the improbability of sufficient improvement to make a working animal useful or to enable a food animal to be fattened. If treatment is to be adopted, sulphate of magnesia and sulphate of soda may be administered along with potassium nitrate or potassium iodide. A generous, laxative, easily digested diet should be administered, together with succulent foods where possible. When the condition is due to parasites in the liver a course of filix mas may effect improvement. In the smaller animals the dropsy should be relieved by puncture of the abdomen, which may have to be repeated at short intervals. The water-supply should be curtailed, and a course of potassium iodide, digitalis, and nux vomica prescribed.

Fatty Degeneration of the Liver

This condition may occur in animals with general fatty degeneration, or it may result from the absorption of certain poisons from the alimentary tract, either bacterial in origin or chemical such as arsenic and phosphorus. In these cases the substance of the liver cells becomes destroyed and in part converted into fat.

A condition of *fatty infiltration* may also occur in which there is no destruction of liver cells, but there is an accumulation of fat which is stored up in the liver cells. This is due to excessive feeding, and is rarely a serious condition.

Symptoms.—The symptoms of fatty degeneration of the liver are very indefinite. They merely consist of interference with digestion, and are associated with general depression, lassitude, and inability to do prolonged work or to undergo any great exertion. Constipation is frequently present, and the fæces are mucus-coated and offensive in odour. An important feature is the danger of rupture of the liver, which may occur as the result of a fall or other injury.

Treatment.—The condition is an incurable one, and also very difficult to diagnose. As a rule, indigestion is diagnosed and the corresponding treatment for indigestion is adopted.

Amyloid Disease of the Liver; Waxy Liver; Lardaceous Disease of the Liver

This condition may arise as the result of the existence of certain chronic bacterial affections such as tuberculosis, chronic suppuration in any part of the body, chronic disease of the serous membranes. It has also been said to occur in contagious pneumonia and chronic kidney affections.

Symptoms.—These are very indefinite, and if any other disease has been in existence first amyloid liver may not even be suspected. Symptoms are very similar to those of fatty degeneration of the liver, and are mainly associated with the absence of bile from the intestines. As in the case of fatty degeneration, the chief danger is the possibility of rupture of the liver and death from internal hæmorrhage. In some cases intermittent jaundice has been noted with occasional attacks of colic. The writer has met with amyloid liver in a horse which had not been previously suspected of being ill. The animal had been in regular work right up to the day of his death, and was found dead the following morning from rupture of the liver, which was found to be affected with amyloid degeneration and to weigh 65 lbs. No other lesions were discoverable.

Treatment.—The same remarks apply in this condition as in the case of fatty degeneration of the liver. No serviceable curative treatment is known even if the condition could be definitely diagnosed.

Rupture of the Liver

Rupture of the liver may be caused by external violence, such as a fall, kick, severe struggling, fractured ribs, or the presence of a foreign body which may have been swallowed. In the majority of cases rupture of the liver is preceded by fatty or amyloid degeneration. It may occasionally be secondary to abscess of the liver, acute septicæmia, or the presence of tumours or parasites in the liver. The degree of rupture varies, and may consist of small hæmorrhages which can be contained and spontaneously controlled by the capsule of the liver. In extensive rupture the capsule of the liver is also torn and severe internal hæmorrhage occurs.

Symptoms.—In the case of small hæmorrhages of the liver no appreciable symptoms are produced. In cases where the rupture is larger but the capsule remains intact the patient shows evidence of subacute abdominal pain, the mucous membranes become pallid and yellowish, the pulse weak, and the extremities cold. There are slight constipation, stiffness of movement, and tenderness on pressure over the area of the liver. There is also said to be lameness of the right fore limb. This condition may exist for several hours or several weeks, and is usually ultimately followed by muscular tremors and internal hæmorrhage and death. In cases of extensive rupture the symptoms are those of internal hæmorrhage. The pulse gets gradually weaker until it becomes almost imperceptible (a so-called running-down pulse), mucous membranes very pallid, the body surface and extremities cold, there may be sighing and yawning, sometimes cold patchy sweats. The patient staggers and falls and the internal temperature becomes low, and this is quickly followed by death.

Treatment.—Treatment may be adopted in the case of small ruptures. The animal must be put in a warm comfortable place and absolute quietness insisted upon. Rugs and bandages or clothing should be applied. All exertion must be carefully avoided. Medicinally, internal styptics such as ergotin, adrenalin, and perchloride of iron have been respectively recommended, but they are of doubtful service. The two former increase blood pressure, and it is probable that they would therefore increase the danger of hæmorrhage from the rupture. On the other hand, heart sedatives such as antipyrin and amyl nitrite which dilate cutaneous vessels may be administered in order to reduce the pressure. Any constipation should be relieved by enemata. The intravenous or hypodermic injections of small quantities of normal saline solution have been recommended, but they are of doubtful service. In the case of food animals slaughter is best, and the carcase should be carefully examined. If it is emaciated and the organs affected with fatty degeneration it should be condemned for food. If the rupture is due to accidental causes and the carcase is in good condition and otherwise fit it may be passed for food.

Bacterial Necrosis of the Liver

This condition, which has also been called *nodular necrosis of the liver*, is due to the necrosis bacillus which gains access to the liver from the alimentary tract by way of the portal blood.

It is fairly commonly met with in cattle, but it has been described as occurring in rare cases in the sheep, horse, pig, and dog. Areas of so-called coagulative necrosis are set up which extend outwardly and attain the size of walnuts. They are well defined in outline and dirty white or yellowish white in colour with a rather dry texture on cutting. The lobulation of the liver can usually be recognized on the cut surfaces. There is sometimes a surrounding area of inflammatory reaction, and in other cases the diseased part may become converted into a substance like thick tough pus around which a fibrous capsule is developed. On rare occasions the nodules undergo calcification.

Symptoms.—These are rarely sufficient to afford a basis for positive diagnosis during life, and the condition is not infrequently met with in the abattoir without its existence having been suspected. If the lesions are small in extent there may be no symptoms produced. In the majority of cases symptoms resemble those produced in suppurative hepatitis or portal congestion, with the exception that dropsy is not so likely to occur as in the latter case. The bowels are irregular, the mucous membranes yellowish and anæmic, and there is loss of flesh and general wasting. In acute rapidly developing cases there is a high fever, with blowing respirations and indication of pain in the region of the liver. Acute jaundice may arise, the patient becomes extremely depressed and weak, the weakness almost amounting to paralysis.

Treatment.—Owing to the impossibility of making a definite diagnosis no special measures can be recommended. In cases where the condition is suspected slaughter is the best procedure; otherwise a course of potassium iodide and quinine may be adopted, but with doubtful results.

Tumours of the Liver

These are not a very common occurrence, but are more frequently met with in the dog than in other species.

In the case of *cancers* (*carcinoma* and *sarcoma*) the symptoms set up are those of gradual wasting and loss of flesh. The liver becomes enlarged, and in the dog and cat can generally be felt on palpation behind the ribs. Dropsy of the abdomen is commonly met with, and the fluid is frequently blood-tinged, and rapidly collects again after having been removed by tapping. The condition may be confused with cirrhosis of the liver and tuberculosis of the liver. There is no satisfactory treatment.

Cavernous Angiomata of the Liver.—These are fairly common in the liver of the ox. They are usually multiple and look almost like blood spots. They do not give rise to any symptoms during life, and are usually only discovered in the abattoir. Cases have also been recorded in the liver of the horse, the dog, and the cat, but they are of extremely rare occurrence. The chief importance lies in the danger of rupture, which may terminate fatally.

Other tumour-like growths are sometimes met with in the liver of *tuberculous* or *actinomycotic* origin. In the case of tuberculosis it may be met with in all the domesticated animals except sheep, and in the case of actinomycosis in cattle.

Large *echinococcus cysts* are also frequently found in the liver, especially in cattle and pigs, producing a tumour-like appearance. They are readily distinguished by puncturing them, when the contained liquid, which is present under tense pressure, spurts out a considerable distance.

Parasites of the Liver

The liver is frequently the habitat of parasites, and this is probably owing to the facility with which they gain access from the intestines. The two main channels of entrance are (*a*) the common bile duct which opens directly from the intestines, and (*b*) the portal vein which leads direct from the intestinal wall to the liver.

The effects of parasites in the liver depend upon the number present and also upon the position. In the former case the liver tissue may become destroyed by pressure, etc., and in other cases the bile ducts may become occluded, setting up jaundice; in each case interference with general nutrition may arise. In long-standing cases cirrhosis is set up. The common parasites met with in this organ include the various flukes (distomes, amphistomes, and bilharzia); the cystic stages of various tapeworms, such as the echinococcus, or embryos migrating through the liver to the abdominal cavity, such as *Cysticercus tenuicollis* and *C. pisiformis*; the *Linguatula denticulata*; *Porocephalus armillatus*; larvæ of sclerostomes; and occasionally ascarides. To these may be added *Coccidium oviforme.*

Distomatosis

(*Fascioliasis; Distomiasis; Liver Fluke Disease*)

This is a disease of the liver set up by trematode worms, *Distoma hepaticum*, *D. lanceolatum*, *D. gigantica*, and *D. magna.*

Flukes may be met with in the liver of many animals, but rarely set up symptoms in other than sheep and cattle. In sheep, liver rot is frequently responsible for serious losses, and is also known to farmers and butchers under such names as "caud" and "cothe."

The *Distoma hepaticum* (*Fasciola hepatica*) has a flattened leaf-like body about an inch long and half an inch wide—broad and rounded in front with a small conical projecting head or neck, and becoming narrower behind with a blunt end. It is dirty white or pale brown in colour. It has a world-wide distribution.

D. gigantica, the common liver fluke in Africa, is long and band-like, being about 1½ inches to 2 inches long and only ¼ inch broad.

D. magna occurs in the United States and is about an inch long and almost as broad.

D. lanceolatum is a very much smaller fluke than the *Distoma hepaticum*, and is about ⅓ inch long and ⅛ inch broad, and is lanceolate in shape as its name implies.

These flukes affect young animals more than adults and thus cause more trouble in lambs and sheep up to two years old, and in cattle up to two or two and a half years old, although they may be met with in large numbers in adults. Conditions predisposing to infestation of these parasites include dampness of the pastures and in general marshy districts. Animals that are housed and fed in stalls are rarely infected except they are given green foods from infected pastures. Infestation occurs principally during the wet seasons, particularly in the autumn, but may occur at any time of the year. A very short time, a few hours only, on a damp infected pasture is quite sufficient to infect a previously healthy sheep with serious results. Infestation of the pastures may also be maintained by rabbits.

Life-history of the Distoma hepaticum.—This is somewhat complicated, but very important. The adult flukes in the bile ducts become mature and deposit their eggs, which are passed into the intestines in enormous numbers chiefly in the spring and early summer. They are passed out with the fæces, and under suitable conditions of temperature and moisture hatch out embryos in from three to six weeks. The embryo is called the *planula* or *miracedium* and possesses cilia and the power of spontaneous movement. If this embryo does not come into contact with the necessary intermediate host it soon perishes. On the other hand, if it is brought into contact with its intermediate host, which is a fresh-water snail, *Limnœa truncatula*, it penetrates it by means of its boring apparatus, and develops into a small cyst called a *sporocyst*. The snail possesses a small spiral conical shell, is ashy grey in colour, about half an inch long and a quarter of an inch in diameter, and with a very wide distribution throughout the world. It is said, however, not to exist in Australia, so that some other similar snail must be capable of harbouring this parasite. In the sporocyst may be developed other sporocysts, and within them small projections, from six to eight, which are called *redia*. These become set free and migrate to the liver of the snail and undergo further development into *cercariœ*. After a while these escape from the body of the host, swim about in the water, and then attach themselves to a leaf or plant. In this situation they develop a mucous coating or *cocoon*, and in this encysted form may be eaten by a susceptible animal such as a sheep or an ox. These embryo flukes are released in the stomach and small intestines and migrate into the liver, where they develop into adult flukes and thus recommence the cycle. The full period of development takes from ten to twelve weeks.

Symptoms of Liver Rot in Sheep.—When the parasites are few in number they may give rise to no symptoms by which their presence can even be suspected, and in some instances sheep with a numerous infestation of the liver remain in a fat and prime condition, as may frequently be seen in the abattoirs. Young sheep are more likely to develop symptoms than older ones. In the early stages of infestation in moderate numbers it is even suggested that in consequence of the stimulation of the secretions of bile there may be an improvement in nutrition and general condition, the sheep actually appearing to be thriving better. In from two to three weeks after infestation symptoms generally supervene, although in some cases several months may elapse. Experienced shepherds are said to be able to distinguish affected animals in the very early stages by manipulating the loins, the muscles of which become soft and flabby, and there is also said to be produced a feeling as of slight emphysema under the skin—a sort of " rippling." In the late autumn or early winter, September, October, and November, some loss of condition may be observed, the sheep becoming dull and less active, the appetite begins to fail, rumination is irregular, the conjunctiva becomes pallid and watery, the fleece becomes harsh, dry, and brittle. As the winter advances the symptoms become more pronounced, the emaciation more obvious. Some degree of fever arises, and the mucous membranes may become somewhat bile-stained, but pronounced jaundice is rare. The bowels are irregular, and eggs of the parasites may be found in the fæces. There is a tendency to œdema of the sub-maxillary space and the body cavities, particularly the peritoneum. The great muscular wasting, particularly along the back producing prominent vertebral spines, and the œdema of the jaw and abdomen are often summarised by the shepherd into the phrase " pot-belly, bottle-jaw, and razor-back." Pregnant ewes may abort, and the milk of ewes with lamb becomes thin and watery and lacking in nourishment so that the lambs may die from malnutrition. The weakness and anæmia increase and death may follow from exhaustion.

In older sheep recovery may occur after a mild attack, but as a general rule prognosis in sheep is unfavourable.

Symptoms in Cattle, Swine, and Horses.—In animals other than the sheep the disease runs a less severe course. There are loss of condition, intermittent appetite, and irregular bowels with pallid and watery mucous membrane, occasionally yellowish tinged. In young cattle death may occur as in the case of sheep from exhaustion, but in adults recovery is comparatively frequent. Positive diagnosis is made by the discovery of eggs of the distoma in the fæces.

Post-mortem Appearances.—The liver in both sheep and cattle is considerably enlarged and may show peritoneal adhesions. It is much firmer and more dense than normal, but the cirrhosis is never uniformly distributed. The bile ducts are thickened and surrounded by increased connective tissue, which makes their presence very obvious. On cutting into it the liver tissue is tough, and the bile ducts are dilated, their contents catarrhal and often greenish-brown in colour. Parasites may be pressed out from the ducts. Sometimes abscesses are produced, and at other times calcification of the bile ducts may occur. Sometimes the gall-bladder is found to contain both parasites and their eggs. In addition to the changes in the liver there is often a quantity of liquid present in the chest and abdomen. There is a marked reduction or complete absence of fat, with general signs of anæmia. Sometimes flukes may be found in other obstructions such as the lungs, muscular tissue, and peritoneum.

Treatment.—Curative treatment in advanced cases is not hopeful, but in early cases the administration of ethereal extract of filix mas is reported as having produced very favourable results. This drug is specially credited in possessing parasiticidal action on parasites in the liver. For sheep it should be administered in ʒi. doses on five successive mornings in Ol. lini ℥i. For cattle the dose on five successive mornings is from ʒiii. to ℥i. in Ol. lini ℥ii. to ℥iv. according to the size of the animal. In both cattle and sheep food should be withheld for two hours after dosing. Other agents which have been recommended include common salt, iron salts, naphthalin, turpentine or herbs containing it such as pine tops, juniper leaves, etc. The following combination has also been recommended by Woodruff :

Ferrous sulphate ℥i.
Sodium chloride ℥ii.
Juniper berries ℥x.
Powdered gentian root lb. i.
Oatmeal or barley meal lbs. xxiv.

Of this mixture half a pound should be given to each sheep daily. Animals must be given a generous allowance of highly nutritious foods, and must be taken up off the pastures where they are being infested and also disseminating ova.

Preventive Measures.—Prevention is far more important than curative treatment, and more likely to give satisfactory results. The main principle consists in the provision of conditions which are unsuitable for the development of the fluke and of its intermediate host, the snail. Infested pastures should be well drained and generously dressed with salt (5 cwts. to the acre) or with gas-lime; ditches and stagnant water should be fenced off. Badly infested pastures should be ploughed up. Over-stocking predisposes to infestation with all parasitic affections, and therefore should be carefully avoided in districts where fluke disease is known to exist. Badly affected animals should be slaughtered and their livers destroyed. It must also be borne in mind that rabbits and hares may act as ultimate hosts of the fluke and thus propagate the disease in the absence of sheep. Obviously, therefore, these animals should be exterminated in infested districts. It is well wherever possible to leave pastures vacant of ruminants for at least one season or two if it can be managed.

Paramphistoma siamense.—This is the only species of amphistome normally found in the liver (amphistomes usually inhabit the intestines). It has been found in large numbers in the bile ducts of calves in Siam and the Malay States. It is about the size of a barley grain. Nothing is known of any symptoms set up by it.

Flukes of the Dog and Cat.—In addition to *D. lanceolatum* a number of other distomes are common in dogs and cats in some parts of the world, though not in the British Isles.

Opisthorchis felineus is frequently met with in the cat in Eastern Prussia.

O. pseudofelineus occurs in cats in North America.

O. noverca infests dogs in India.

Clonorchis sinensis, a parasite of man in China and Japan, is also common there in dogs and cats.

Dicrocœlium concinnum is common in cats in British Guiana, and produces marked "pipey livers."

Metorchis truncatus is the smallest of the liver flukes of the dog and cat, being about $\frac{1}{12}$th inch long and $\frac{1}{30}$th inch wide. It occurs in Central Europe.

There are no clinical data so far recorded concerning these flukes.

Bilharziosis.—The immature stages of various species of Bilharzia worm inhabit the portal veins of the liver. When numerous they may give rise to enlargement and cirrhosis of the liver with the development of white plaques of fibrous connective tissue surrounding the portal vessels.

The lesions result chiefly from the irritation by the eggs laid by the parasite, and partly to toxins they elaborate.

Clinically, the principal symptoms are those of dysentery, due to a similar deposition of eggs in the finer branches of the portal veins in the walls of the intestine, especially the rectum.

The parasites are easily collected by incising the portal vein at its entrance to the liver. The blood forced out by jerking is caught in a large basin containing water. After allowing it to stand about half an hour the liquid is poured off and the worms are left in the sediment.

The species include :

Bilharzia japonica, affecting cattle, dogs, cats, and man. China and Japan.
B. bovis (*B. crassa*), affecting cattle and sheep. Egypt, Soudan, Nyasaland, Sicily.
B. indica, affecting horses. India.
B. bomfordi, affecting sheep. India.

Bladder-worm Disease of the Liver.—The cystic stages of several tapeworms of the dog and cat may be met with in the liver of various animals. The chief ones are :

Echinococcus veterinorum (*E. polymorphus*), cystic stage of the *Tœnia echinococcus*.
Echinococcus multilocularis (*E. alveolaris*), cystic stage of the *T. echinococcus* var. *alveolaris*.
Cysticercus tenuicollis, cystic stage of the *T. marginata*.
Cysticercus pisiformis, cystic stage of the *T. serrata*.

The two former set up a condition known as echinococcosis, while the two latter set up cysticercosis of the liver.

Echinococcosis.—Echinococcus cysts are frequently met with in the liver of cattle, sheep, and swine, and less frequently in the horse and dog. They may be present in large numbers or they may be few in number. The cysts vary from the size of a pea up to the size of a cocoa-nut. In bad cases the liver becomes enormously enlarged. Affected livers have been met with weighing over 1½ cwts. in cattle, while in pigs they may reach 40 or 50 lbs. in weight.

Symptoms.—If the cysts are present in small numbers few or no symptoms are set up; when present in large numbers they cause interference with the functions of the liver, and set up some degree of indigestion, irregular appetite, and irregular bowels. In cattle there is frequently tympany. When the cyst presses upon the bile ducts it may partially occlude them, setting up some degree of jaundice, which, however, is rarely very pronounced. There is generally loss of condition and wasting. When the liver is very markedly enlarged it may be felt on manipulation of the abdominal wall immediately behind the last rib in the lower half of the abdomen. In such cases there may be some mechanical interference with respiration. Definite diagnosis of echinococcosis is, however, rarely possible, although extensive invasion of the liver may prove fatal owing to the destruction of liver tissue which it entails.

Post-mortem Appearances.—Cysts of *Echinococcus veterinorum* of the liver are, as a rule, very obvious and easily seen. They cause rounded prominences of various sizes, and cause distortion of the liver if very numerous. The cysts are very tensely filled with clear, watery liquid which is enclosed in a dense fibrous wall. When the cysts are cut the fluid spurts out a considerable distance. Inside the cyst may be found a number of other cysts (daughter and grand-daughter cysts), and these may contain small projections (scolices) which are potential heads of future tapeworms, and which, if ingested by a dog, develop into *T. echinococcus* in his intestines. Some cysts, however, are barren (acephalous). When the cysts are not numerous they may perish and undergo degeneration. The fluid is absorbed and the cyst shrinks to quite a small size, and may undergo calcification, and in this condition may be mistaken for tuberculous lesions. They may be distinguished, however, by the fact that they can be shelled out with the point of a knife, and also by the fact that the hepatic lymphatic glands are not involved. Microscopic examination also reveals the presence of hooklets from the scolices, and a lamellated appearance of the wall of the parasite.

The cyst of the *Echinococcus multilocularis* differs in appearance from the former. It presents more tumour-like growths of fairly firm consistence, and when cut into shows a considerable number of cysts of small size. Old cysts on section frequently show calcification of the central portion, the outer part being undegenerated but containing small cysts.

Treatment.—No curative treatment is possible, but prophylaxis may be practised since the disease is caused by the ingestion of eggs of the tapeworm harboured by dogs. Care should be taken to prevent the soiling of food or water with the excreta of dogs. If the condition already exists on the premises it is possible that dogs are already infested, and they should be accordingly treated for tapeworms. Care must also be taken that any organs or tissues of animals affected with echinococcus cysts should be destroyed and not be given to dogs.

Cysticercosis.—The commonest cause of cysticercosis of the liver is the *Cysticercus tenuicollis*, which is met with principally in pigs and sheep and young cattle. *Cysticercus pisiformis* is also frequently met with in the liver of rabbits and hares. Infestation is due to the ingestion of eggs of the *T. marginata* and the *T. serrata* of the dog respectively. The embryos liberated from these ova migrate through the intestinal wall and are carried by the portal vein to the liver, through which they pass on their way to their predilection seat in the peritoneal cavity. Channels or tracks are left marking the routes taken by the parasites.

Symptoms.—When cysticerci are very numerous they may set up a serious illness and may prove fatal, death being preceded by marked dulness and depression, rapidly developing debility, and a staggering gait. These symptoms may be set up in a week or ten days after the ingestion of large numbers of eggs. In the majority of cases, however, the invasion is more gradual and the symptoms are less pronounced, consisting of dulness and weakness, loss of appetite, and irregular bowels, great loss of condition, and may prove fatal. When the parasites are few in number no symptoms are set up by which it can be diagnosed during life.

Post-mortem Appearances.—In acute stages the liver may be enlarged, but in mild infection there is no alteration in the size of the liver. The organ may be spotted, blackish red or greyish brown. In the spots may be seen young cysts up to a quarter of an inch in width. There may also be seen channels or burrows marking the tracks along which the parasites have passed. In rare cases acute peritonitis with blood-stained liquid may be met with. In older cases in ruminants and pigs some of the cysts may be seen attached to the peritoneum covering the liver, and are then much enlarged and possess an elongated neck. In rabbits and hares after leaving the liver the cysts are seen attached to the omentum and mesentery in considerable numbers, and attain the size of a marrowfat pea. Each cyst possesses one scolex.

Treatment.—No successful curative treatment is known, but prophylaxis may be practised by the administration of tæniafuges to the dogs, and the prevention of contamination of food and water with dogs' excreta.

Lingatulidæ.—These are parasitic arachnida, with ringed and elongated worm-like bodies. There are two genera, *Linguatula* (or *Pentastoma*) and *Porocephalus.*

The adult stage of the former are found in the nasal chambers of dogs, and less frequently in the horse, sheep, goat, and cat (see p. 399).

The larvæ, up to ½ inch in length, are met with encysted in the liver just below Glisson's capsule in cattle, sheep, rabbits, and horses, and rarely in cats and dogs.

The adult stages of the *Porocephalus* occur in African pythons and other snakes. Their larvæ are found coiled up and encysted in the liver, mesentery, and sometimes lung in cattle, sheep, and man in West Africa.

G. H. W.

DISEASES OF THE PANCREAS

Diseases of the pancreas are rarely met with in veterinary practice, and still more rarely are they diagnosed during life. This is largely owing to the fact that the secretions of the digestive fluid of the pancreas can be replaced by secretions of other organs. In other words, the functions of the pancreatic juice are really duplications of those performed by other secretions. The principal affections met with consist of catarrhal inflammation of the pancreas, suppuration of the pancreas, atrophy, hypertrophy, tumours, and calculus. Inflammation of the pancreas and obstruction of the duct are most frequently produced by extension of catarrh from the small intestines up the pancreatic duct. In other cases they may be due to complications of a general infection.

Symptoms.—The symptoms of interference with the action of the pancreas are principally those of indigestion owing to the arrest of the secretion of pancreatic juice and its absence from the bowel. The principal one, therefore, is some degree of indigestion with irregular appetite and dulness, intermittent tympany, irregular bowels, general loss of condition, and inability to perform prolonged exertion. There is frequently abdominal pain, and in dogs vomiting. The fæces are seen to contain undigested food. The urine frequently contains sugar; in fact, saccharine diabetes is frequently attributed to disease of the pancreas. There is often some jaundice, which is likely to cause confusion of diagnosis. In carnivora the fæces may be oily, a condition which it is sometimes assumed is due to the withholding of the fat-emulsifying constituent (steapsin) of the pancreatic juice. In a case met with by the writer in a dog pronounced fatty stools were passed, the first portion of the stool consisting of greyish fæces and followed by what appeared to be a quantity of oil, which had a very pronounced odour resembling rancid butter. On becoming cold the oil solidified into a substance resembling in appearance a wax candle, and on chemical examination it was found to consist mainly of stearin. When milk or milk products and all foods containing fats were rigorously withheld from the diet, the fatty stools still persisted and the dog continued to waste in condition. This fact would suggest that the presence of fat was not due to the lack of digestion of food constituents. Treatment, which included the administration of pancreatic secretions and extracts, was of no service. The animal was eventually destroyed, and on post-mortem examination being made the pancreas was found to be completely atrophied with nothing left but a little connective tissue to mark its position. Other cases have been recorded in which the pancreas of the horse was very considerably increased in size and indurated.

Treatment.—Owing to the difficulty of diagnosis the treatment is largely experimental. If diseased pancreas is suspected, pancreatic extracts or secretions should be administered, and fresh sheep's pancreas may be fed to affected carnivora. Apart from this, treatment is the same as for indigestion, and consists of a course of general alimentary tonics with attention to diet. Results, however, are not very hopeful.

Parasites of the Pancreas

Eurytrema pancreaticum.—**In cattle** in China enormous numbers of this species are frequently found distending the pancreatic duct. They have only been found on post-mortem examination and no clinical data have been recorded.

The parasite is a distome, nearly related to *D. lanceolatum*, but is somewhat longer and twice as broad.

G. H. W.

DISEASES OF THE SPLEEN

The spleen is the largest of the " ductless glands " and lies in close relationship to the greater curvature of the stomach or in ruminants in a similar relation to the rumen, on the left side of the body.

Externally it is of a bluish-red colour, and internally a dark red, being soft and " fleshy " although not friable in consistency. On the side touching the stomach is the *hilus*, at which the vessels and nerve enter.

The function of this organ has not been definitely determined although it may be considered " the birthplace and cemetery of the red corpuscles of the blood." Certainly also it has some bearing on leucocyte production. Nevertheless the whole organ may be removed without harm to the patient.

In the lower animals spleen diseases are rarely diagnosed except at post-mortem examination. They are of little import except as an aid to diagnosis of specific diseases.

Engorged and Enlarged Spleen.—To be noticeable this must be very considerable as there is great normal variation in size.

It may be due to portal obstruction or even to torsion, in which case the splenic vein is twisted. A common cause of the condition in a typical form is anthrax, where the spleen is ten or fifteen times the normal size in cattle and of a very soft consistency, as the extra bulk is entirely due to blood. If not caused by some rapidly fatal disease the enlargement may become chronic, the consistency becoming firmer as fibrous tissue is formed. Such a condition is common in swine erysipelas and also in Hodgkin's disease. Histological examination shows no abnormality in chronic enlargement.

Various protozoan diseases cause initial enlargement of the spleen, and if a patient dies in the early stages a microscopic examination of a spleen smear may show the causal parasite. Particularly in East Coast fever is a spleen smear of value.

Atrophied Spleen.—Due to venous stasis or torsion. It may lead to necrosis, but there are no symptoms.

Splenitis.—This is usually specific and may be due to diseases like anthrax or to secondary infection from a septic focus. In the latter case multiple abcesses are found. In tuberculosis splenic lesions may be present, the most typical being in the horse.

Traumatic splenitis is rare but may be found in cases where a foreign body has worked through from the stomach. Diagnosis is difficult, for except when due to a specific disease splenitis has no symptoms beyond a degree of debility.

Hæmorrhage.—In cases where hæmorrhage is present there is usually history of a violent blow or accident. General symptoms of internal hæmorrhage are shown; rapidly advancing pallor of mucous membranes being followed by unconsciousness and in small animals perhaps a pendulous appearance of the abdomen. It is invariably fatal.

New Growths.—Sarcomata are the commonest, particularly melanotic sarcomata in aged grey horses. Echinococcus cysts may form in the substance of the organ.

Amyloid (Waxy) Spleen.—In domesticated animals this is an indication of long-standing fever or chronic suppuration. In the horse the spleen is much firmer than normally.

Specific Diseases.—The main diseases producing obvious splenic lesions are anthrax, redwater, tuberculosis, East Coast fever and Hodgkin's disease.

In anthrax, particularly in the ox, the organ is greatly enlarged, soft, and on section black and semi-fluid. Soon after death microscopic examination shows the bacilli in large numbers, but after a day or two these die and disintegrate if the carcase has meanwhile remained unopened.

In tuberculosis, tubercles may be seen. In most animals these are small and numerous, and in the ox usually confined to the peritoneal covering, but in the horse a typical, definitely circumscribed, tough tumour is presented, varying in size up to a cocoa-nut. These are the so-called *lymphadenomata*. Such " tumours " are rarely very numerous.

In redwater the spleen is usually enlarged if death was due to the disease, but never to the extent found in anthrax. Spleen smears may show the piroplasms.

In East Coast Fever the spleen is *not* enlarged, but spleen smears examined microscopically afford the best material for diagnosis.

In Hodgkin's disease the spleen is enlarged and unsoftened, and histological examination shows no abnormality. In conjunction one also has the general enlargement of lymphatic glands to aid diagnosis. Apart from definitely diagnosed Hodgkin's disease it is not uncommon to find in dogs and cats a spleen showing multiple small areas of severe hyperplasia.

S. C. J. B.

DISEASES OF THE PERITONEUM

Peritonitis; Inflammation of the Peritoneum

The peritoneum is the serous membrane of the abdominal cavity. It therefore lines the inner surface of the abdominal wall, the posterior face of the diaphragm, and is reflected by way of the mesentery and omentum over the outer surface of the various abdominal organs.

Peritonitis is due to the invasion of the abdominal cavity by micro-organisms. When their method of entrance is through a wound it is spoken of as *traumatic peritonitis*. The wound may be through the abdominal wall or from the stomach or intestines as in rupture, sometimes preceded by an ulcer; in cattle it may be due to a foreign body such as a nail piercing the rumen or reticulum, in the dog to foreign bodies such as skewers or sharp bones. It may be secondary to castration and also to rupture of the womb or of the bladder. These cases are almost always putrid in character and are fatal. In any species peritonitis is very rarely primary. It is frequently secondary to affections in various parts of the body such as pleurisy, diseased mesenteric lymphatic glands, suppuration of the liver, strangles, invasion from the bowel in consequence of strangulation or twist or calculus in the horse. In cattle the tubercle bacillus is a frequent cause of peritonitis, and to a less common degree also in dogs and cats. Parasites are frequently met with in the peritoneum, but rarely set up any inflammation except in intense infestation with *Cysticercus tenuicollis* in sheep and pigs.

Symptoms.—There is usually a history of some previous affection or injury which may be discovered, and which is followed by the symptoms of peritonitis, coming on either gradually or suddenly. Acute peritonitis is usually a very painful condition, but a cold or so-called quiet peritonitis may exist, causing little or no pain as in tuberculosis of the peritoneum. In acute peritonitis the patient becomes very restless, constantly looking round at the abdomen, and pressure on the abdominal wall causes pain. Horses and cattle paddle with their hind feet, and the former paw with their fore feet. Respirations are increased in number, the chest wall working exaggeratedly, the abdominal wall being fixed. The pulse is quick, frequent, small and wiry; the temperature elevated four to six degrees. There is an indisposition to pass fæces, although in the earlier stages there may be diarrhœa which gives way to more or less obstinate constipation. This may give rise to a varying degree of tympany. In dogs vomiting is frequent. Horses and cattle with peritonitis rarely lie down, but the smaller animals lie down and become prostrate. The patient frequently places himself in a position to stale and then refrains. In some cases there is frequent urination, but at other times retention of urine. As peritonitis progresses, exudation into the peritoneum occurs and the condition becomes less painful. The fluid may be discovered on palpation, and on percussion a trill or vibration may be experienced on the opposite side of the abdomen. This is particularly noticeable in the smaller animals. Diagnosis can be confirmed by an exploratory puncture of the abdomen.

In cases of local or circumscribed peritonitis the pain is less severe and the general symptoms are less pronounced, frequently corresponding only with those of indigestion, but may result in local adhesions. In chronic peritonitis the symptoms are very indefinite. In such cases the patient is often reduced to an asthenic condition with wasting and great debility preceded by irregular bowels and symptoms of indigestion. There may be œdema of the abdominal wall, especially the scrotum and sheath in the horse.

The accumulation of fluid is variable, but an exploratory puncture may be resorted to for positive diagnosis. In horses and cattle the quantity may be anything from two to ten gallons, in dogs up to one or two gallons. In character it may be clear and slightly blood-stained, containing flakes of fibrin. In other cases it is turbid and sometimes of thick consistence, putrid in traumatic cases but almost odourless in others. Tubercular peritonitis in cattle is usually a dry peritonitis, while in dogs and cats it is almost always an exudative peritonitis.

Treatment.—Radical treatment is only possible in the small animals, and consists of performing laparotomy and irrigating the peritoneum first with normal saline solution followed by a mild antiseptic such as eusol or iodine ½ per cent, or chinosol 1-2000. Such methods are obviously impossible in the larger animals. Medicinally the administration of opium or morphia affords very considerable relief by reducing peristaltic action as well as by its anodyne action on the higher nerve centres. Constipating effects, however, must be controlled by the administration of mild oleaginous laxatives in the larger animals and paraffinum liquidum or petroleum emulsion in the smaller animals. The administration of copious hot enemata affords relief and facilitates removal of fæces from the lower bowel. Febrifuges and internal antiseptics must be administered, the best results being obtained from potassium iodide, quinine, salicylates, and carbolic acid. In very painful conditions, if the heart of the patient is strong, tincture of aconite may be given. Stimulants may be necessary, such as ammonia by the mouth or ether which may be given hypodermically. Hot applications or mustard plasters to the abdominal wall also afford relief. When there is fluid present it should be removed by the trocar and cannula, and puncture may have to be repeated on several occasions.

Acute peritonitis is a very serious condition, and especially if it is of a septic character; such cases are almost always fatal in from two to four days. When peritonitis is non-septic the prognosis is rather more favourable. In the majority of cases where recovery occurs, adhesions of the viscera to one another and to the abdominal wall are frequent. These conditions, however, can rarely be diagnosed during life.

Ascites or Dropsy of the Peritoneum

This is a condition in which the abdominal cavity becomes distended with a liquid called *a transudate.* The fluid must be distinguished from inflammatory liquids, which are called *exudates.* The chief differences between the two are:

(*a*) Inflammatory exudates are generally rich in fibrin-forming elements and frequently undergo spontaneous coagulation. Dropsical liquids nearly always remain liquid while in the body.

(*b*) Inflammatory exudate is richer in albumen than dropsical fluids.

(*c*) Inflammatory exudates are usually much richer in corpuscles red and white.

(*d*) Dropsical parts are never hot or painful.

Dropsy of the peritoneum is a common result of various diseases rather than a disease in itself.

Causes.—The principal cause is some interference with the circulation of the vessels of the splanchnic or abdominal area or of the liver in particular This may be due to pressure on the portal vein by tumours or cysts, or may be due to cirrhosis or to regurgitant disease of the right side of the heart, or debility of the left side of the heart, or destructive lung disease. Dropsy of the peritoneum is also met with in cachectic or debilitated patients in advanced stages of wasting diseases such as nephritis and liver rot in sheep. This form is often called *hydræmic ascites.* In dogs and cats the commonest causes of ascites are abdominal tumours and tuberculosis of the peritoneum. The fluid in ascites is generally clear and almost colourless, at other times it is slightly turbid and sometimes faintly yellow. In rare cases it is opalescent, probably due to the presence of blood pigment. If associated with jaundice it is yellow.

Chylous Ascites.—This condition is not infrequently met with in cats and rarely in the dog or other animals. In these cases the liquid is milky in appearance, and if placed in a glass vessel would with difficulty be distinguished from milk. It probably arises from a small puncture of a lacteal or some obstructive disease of mesenteric lymphatic glands.

Symptoms.—The symptoms of ascites are usually slow in development. The effusion takes place gradually, and the abdominal wall accommodates itself to the increase in the abdominal contents. When the fluid becomes copious, the abdomen is swollen and pendulous, and the gradual upward curve of the abdominal wall from the sternum to the pelvis is lost. The flanks become hollow, and as a rule the vertebral spines become prominent. On palpation the abdominal wall is found to be more or less tense, and movement of the liquid can be appreciated. In the small animals such as the dog and cat, gentle percussion on one side of the abdomen causes a wave or trill which can be felt by the other hand placed on the opposite side of the body. Respirations may be laboured owing to the pressure of the fluid on the diaphragm, consequently the patient will often take up a position to relieve pressure as far as possible. In the larger animals this consists of standing with hocks and hind fetlocks partially flexed; in the smaller animals the sitting posture is adopted. The temperature is usually normal or slightly subnormal. The pulse varies according to the different causes of the condition. Diagnosis may be confirmed by an exploratory puncture with the trocar and cannula, and may be assisted by rectal exploration.

Prognosis in ascites is generally unfavourable on account of the fact that in so many cases it is due to some primary incurable affection. In other cases, if taken early, the condition can be cured, while in other cases still it may be palliated and the patient kept in a comparatively comfortable condition for months or even years.

Ascites must be distinguished from peritonitis, dropsy of the uterus, myoma of the uterus, and also pregnancy.

Treatment.—In the majority of cases treatment is only palliative. If the fluid is copious it should be drawn off by tapping with a sterile trocar and cannula (paracentesis abdominis), but as the condition is frequently recurrent it may be necessary to repeat the process with intervals of four or five days or a week. In favourable cases the intervals become longer, while in unfavourable cases the fluid collects with great rapidity and repeated tapping causes intense debility and depression. In persistent cases in the smaller animals such as the dog and cat, laparotomy may be performed and the abdominal cavity irrigated with an antiseptic astringent such as iodine solution (1-300 or 1-500) or a fairly strong solution of chloral hydrate (1-5 or 10). Surgically, an anastomosis may be obtained by producing an adhesion of the omentum with the abdominal wall, thus affording a fresh channel for the draining of the lymph. Medicinally, a laxative should first be given, preferably a saline to draw off fluid by the alimentary tract. This may be followed by diuretics and cardiac tonics, a useful combination being potassium nitrate and digitalis or squills, to which may be added potassium iodide, which being a sialic minimizes thirst, and therefore reduces the intake of water. If the primary cause of the condition is discovered it must be treated accordingly. The diet must be highly nutritious and generous, and the water supply should be curtailed.

Parasites of the Peritoneum

A number of parasites are occasionally found in the peritoneal cavity of various animals, but they rarely or never cause any serious interference with health, and are only discovered on post-mortem examination.

In the horse may be found *Filaria papillosa* (*Setaria equina*), and also occasionally immature forms of intestinal strongyles (*Str. vulgaris*, *Str. equinus*, *Str. edentatus*).

Filaria Papillosa.—This is a long, white worm, male—3 to 4 inches in length with a spiral tail, female—6 to 8 inches long.

It is often found free in the abdominal cavity or the scrotal sack or interlaced to the peritoneum. It is well known in India, Africa, and Southern Europe, but is unknown in the British Isles except in imported animals. It gives rise to no symptoms.

In ruminants the parasite most commonly met with is the *Cysticercus tenuicollis*, which, in the majority of cases, causes no symptoms, but occasionally, if in excessive numbers, may set up peritonitis.

Others occasionally met with are the *Filaria labiatopapillosa* (*Setaria labiatopapillosa*, *F. cervina*) throughout the tropics, and *Linguatula lanceolata*.

In pigs the *Cysticercus tenuicollis* is the most common. There may also be found *Linguatula lanceolata*, *Stephanurus dentatus*, and *Setaria bernardi*.

In dogs and cats parasites are rarely met with in the peritoneal cavity, but occasionally the following have been met with, *echinococci* and *Linguatula denticulata*. In rare cases *ascarides* have also been found. Several instances of unidentified *cysts* have also been recorded by Sendrail and Guille.

Stephanurus dentatus (*Sclerostoma pinguicola*).—This parasite is a nematode worm closely related to the sclerostomines. It is somewhat sturdy; the male averages about 1 inch in length, and the female about 1½ inches. It has been met with in North and South America, and in Australia, but is not known to occur in Europe. It is found in small cysts in the fat surrounding the abdominal viscera, particularly in the neighbourhood of the kidneys. It has also been met with in the kidney substance, and in the liver, and less frequently in the spinal cord.

This parasite has been described as being responsible for a variety of symptoms such as stiffness and paralysis of the hind limbs, and also general wasting and signs of abdominal affection resembling swine fever. In some outbreaks the record of symptoms so closely resembles the latter disease that it is probable that the real cause of the epizoötic was actually swine fever, and that the pigs were simply affected coincidently with *Stephanurus dentatus*. On the other hand, the parasite is frequently met with in swine without having produced any appreciable symptoms, and it is not to be wondered at that it is found in many pigs dead of other diseases.

A positive diagnosis may be made in some cases by the discovery of the eggs of the parasite in the urine. G. H. W.

DISEASES OF THE URINARY ORGANS

Normal Urine

In the horse the urine is slightly turbid when passed, becoming more turbid after it stands a while. In reaction it is alkaline, due to the presence of carbonate of potassium. It is said, however, that in horses fed entirely on oats the urine may become acid in reaction due to the presence of acid phosphates. The normal specific gravity averages 1036 (1020 - 1050). The quantity passed by the horse varies according to his diet and the time of the year. It is increased in winter and at rest, and diminished in hot weather and at work. The consistence of the urine of the horse is frequently more or less oily, which is probably due to active secretion of mucous glands in the pelvis of the kidney and the bladder.

In ruminants the urine is generally clear and yellowish in colour and alkaline in reaction. The quantity depends largely upon the nature of the food, and since the food of ruminants is usually succulent the amount of urine passed is considerably greater than in the horse. In cattle the amount may be from twenty or thirty pints per day, in sheep from half a pint to one and a half pints. The specific gravity is lower than in the horse; in cattle it averages 1016, and in sheep about 1010.

In the pig the urine is generally clear, with a specific gravity averaging 1020. The reaction depends upon the character of his food at the time. If the food is mainly herbaceous the reaction is alkaline, but if the food is of animal origin the reaction of the urine is acid.

In the dog and cat the reaction corresponds with that of the pig, namely acid on a carnivorous diet and alkaline if on herbaceous diet. The specific gravity depends on the diet and varies from 1016 to 1060. The urine is usually transparent and pale yellow in colour. The amount of urine passed is about one half the fluid ingesta.

The *specific gravity of urine* is altered by the proportions of solids and water present in the urine. If the amount of water excreted is increased in greater proportion than the solids the specific gravity is reduced, and *vice versa*; thus an animal may discharge several times the normal quantity of solids in his urine, and yet the specific gravity may be lower than normal owing to the relatively larger increase in the amount of water discharged at the same time. This condition is met with in polyuria (diabetes insipidus). The specific gravity is increased in albuminuria, diabetes mellitus, in hæmorrhage in the urinary tract, also during early convalescence after febrile diseases.

The *colour* of urine is largely affected by the relative amount of water passed, and by the condition of the liver and intestines. An obstruction to the intestines or to the bile ducts raises the colour of the urine. Any disease of the blood causing destruction of red blood corpuscles in quantity causes a red colour in the urine due to the presence of free hæmoglobin. The urine may also be pigmented red in horses by feeding excessively on red carrots and also upon pea straw. These two latter conditions may readily be mistaken for a pathological condition, but apparently they cause no serious trouble. The presence of blood from any part of the urinary tract also renders the urine red. Certain drugs make the urine darker in colour than normal, such as carbolic acid and rhubarb, while others cause the urine to be lighter in colour than normal, such as potassium nitrate. The urine may be rendered turbid by the presence of mucus, salts, pus, or other inflammatory exudates. The chief salts responsible for turbidity are phosphates, carbonates, and oxalates. When the turbidity is due to phosphates and carbonates it is reduced by heating, but the addition of an acid causes effervescence with carbonates and not with phosphates.

The odour of urine varies according to the diet. It may become offensive in cystitis, and may also smell of ammonia owing to decomposition of urea due to the invasion of the bladder with organisms.

Albuminuria

Normal urine should not contain albumen except when an abundant egg diet has been taken or administered. The presence of albumen in the urine usually indicates some structural alterations of the kidney substance or the bladder (nephritis and cystitis), or chronic venous congestion of the kidney. Albumen in the urine may also arise from the generative organs in the male, particularly the prostate in prostatitis in the dog. This condition is sometimes spoken of as spurious albuminuria since the albumen does not arise from the urinary tract proper. Albuminuria may also occur in certain forms of poisoning without structural alterations, as in poisoning by carbonic acid gas, phosphorus, lead, mercury, and arsenic. It may also be present in some specific diseases such as tetanus. If carnivorous animals are kept on a diet entirely free from salt, a form of albumen may be present in the urine, and in some forms of indigestion. It is also said that albuminuria may occur after great exertions as the result of fatigue.

When albuminuria is due to disease of the urinary tract the albumen may be present in the form of blood, pus, epithelium, serum albumen, and serum globulin. When a lesion of the wall of the tubules allows albumen to escape from the blood it may coagulate there and form casts, so-called hyaline casts. Epithelial casts and blood-casts may also be met with. Chronic albuminuria is frequently known as Bright's disease, and is one of the symptoms of chronic nephritis.

Symptoms of Albuminuria.—Albuminuria itself is in reality a symptom of some pathological condition, as already indicated. When it exists for a prolonged period it causes very great exhaustion, debility, emaciation, and anæmia, and may prove fatal. Definite diagnosis, however, is made by examination of the urine, chemically and microscopically.

Tests for Albumen in the Urine. (1) *Heat Test.*—Some of the suspected urine is poured into a test-tube to a depth of about two inches, and is slightly acidulated with acetic acid. The upper part of the liquid is then boiled in a slightly inclined tube over the Bunsen burner. If albumen is present it will be precipitated, rendering the fluid turbid or producing flakes of albumen.

(2) *The Cold Nitric Acid Test.*—Strong nitric acid is first poured into a test-tube. An equal volume of the urine to be tested is then allowed to trickle down the inside of the tube so as to remain on the surface of the acid. If albumen is present it becomes coagulated at the point of junction of the two liquids, and may present the appearance of a white haze or a distinct opaque white ring, according to the amount of albumen present.

(3) *Esbach's Test.*—Esbach's reagent consists of 10 grms. picric acid and 20 grms. of citric acid dissolved in 1 litre of water. This reagent precipitates albumen in urine in the form of small flakes, rendering it turbid. This is the reagent employed in estimating the quantity of albumen by means of the albuminometer. It also precipitates creatinin, uric acid, and peptones.

(4) *The Ferrocyanide Test.*—The urine is first acidulated with acetic acid in a test-tube, and to it is added a 10 per cent solution of potassium ferrocyanide. If albumen is present a white precipitate is produced, varying from a haze to flakiness, depending upon the amount of albumen present.

Microscopic examination of the urine may be resorted to with a view to determining the origin of the albumen. When the albumen originates from the kidney there may be also present casts—epithelial, hyaline, or blood-casts. The character of epithelium may also be recognized. The capsules and glomerules have a flattened epithelium; uriniferous tubules have spheroidal, columnar, or rodded epithelium; the renal pelvis and ureters have squamous epithelium; the bladder has squamous epithelium also, but in cystitis the cells are larger and more numerous than when they arise from the former situation. The epithelial cells in urine may, however, become so altered in some cases as to be difficult or impossible of recognition. When the albumen is of prostatic origin the urine is turbid as the result of prostatic fluid. This may be detected by the precipitation of long, needle-shaped crystals on the addition of a little ammonium phosphate. In such cases, if the urinary tract is not involved the amount of albumen present is generally small.

Treatment.—As albuminuria is really only a symptom of some pathological condition which may vary from case to case, it is obvious that treatment will depend entirely upon the primary condition existing, hence the necessity for careful diagnosis. In any case, however, owing to the fact that the condition is associated with great loss of flesh special attention must be paid to the diet, and generous supply of nutritives should be given. A very useful combination in such cases consists of cod-liver oil, liquid extract of malt, and phosphate of iron. The patient must also be kept warm and free from exposure, and prolonged or violent exertion must be avoided.

Hæmaturia; Bloody Urine

This is a condition of blood in the urine, and may result from hæmorrhage in any part of the urinary tract, renal tubules, renal pelvis, ureters, bladder, or the urethra. In this condition the total constituents of blood are found in the urine. It must thus be distinguished from hæmoglobinuria, in which urine is coloured red from the presence of *free* hæmoglobin which has been expelled from the blood in hæmoglobinæmia due to the breaking down of large numbers of red corpuscles as in piroplasmosis, and in azoturia of the horse.

Hæmaturia may occur in the course of acute congestion of the kidney, acute nephritis, renal calculus, cystitis, and cystic calculus and urethritis. It may occur also in purpura hæmorrhagica and in poisoning by turpentine and cantharides (due to nephritis).

When the hæmorrhage is into the renal tubules, the blood is present in small blood-casts, and urine containing them is diffusely red on being passed, but on standing in a vessel the casts become sedimented. Renal casts are microscopic in size and elongated or cylindrical, being moulded in the tubules.

When the hæmorrhage is into the pelvis of the kidney the blood may coagulate in the pelvis or the ureter. The urine in such cases may be stained red throughout, and may also contain a cast or mould of the pelvis or ureter.

When the hæmorrhage is into the bladder the blood appears to sink to the fundus, and there coagulates, to be expelled in irregular clots during micturition with the latter portion of the urine discharged, that is, when the bladder is terminating its contraction. There may be some diffuse staining of the bulk of urine. The hæmorrhage in this case can be distinguished from renal hæmorrhage by the absence of tubular blood-casts.

When the hæmorrhage is into the urethra there may be some dribbling, or a drip of blood escaping between the acts of micturition; the first portion of urine passed contains blood, while the bulk of the urine is clear until it comes to the last jet expelled by contraction of the accelerator urinæ muscle, which is usually blood-stained.

Treatment.—This will depend on the origin of the blood. A course of urinary astringents should be given such as balsams, tincture of buchu, and copaiva. The so-called internal styptics, as ergot and adrenalin, are contraindicated for administration by the mouth or hypodermically, as they raise blood pressure and are likely to increase the hæmorrhage. If the hæmorrhage, however, is in the bladder or urethra the local injection of adrenalin may check the hæmorrhage. A course of hæmatinics should also be given in the attempt to compensate to some degree for the loss of blood. A very useful combination for this purpose consists of cod-liver oil, liquid extract of malt, and phosphate of iron. Belladonna and hyoscyamus, together with their alkaloids, although so useful in many urinary affections, should be carefully avoided in hæmaturia, as they generally aggravate or increase the hæmorrhage in the urinary tract by specifically dilating the renal arterioles.

Chronic Hæmaturia in Cattle

A peculiar form of chronic hæmaturia occurs as a definite disease in cattle. It is a widespread affection occurring on the Continent of Europe, in North America, and also in Australia. The blood originates from the bladder, and the condition is seen principally towards the end of winter after the long period of dry feeding, and more particularly in old cattle, the average age being about six years.

The cause of the condition is unknown. It has been suggested, however, that it is due to oxalic acid eaten in various plants, since calcium oxalate crystals are usually found in considerable quantities in the urine of affected cattle. The administration of oxalic acid daily for a considerable period over two years produced hæmaturia and weakness, but at other times similar experimental administration failed to produce the condition. Other asserted causes are insufficient food, irritant poisons, and infection with either bacteria, coccidia or various worms. Streptococci have been found in the bladder and also in the kidneys of affected animals, but as the urine is usually sterile in the early stages of the disease the invasion of the streptococci is thought to be secondary. In British Columbia the disease is confined almost entirely to the lands bordering on the Pacific.

Symptoms.—In the early stages the animals show no systemic symptoms. All that is observed is abnormally frequent urination and the passage of a little blood with the last portion of urine. This may last for a few days and recur a few weeks later, and may go on in this way for a year or two, the animals being apparently in good health with the exception of the passage of bloody urine. In the later stages, which may be a year or two after the first symptoms have been observed, the animals develop a depraved appetite, become emaciated and anæmic, and diarrhœa may supervene. In some cases incontinence of urine is set up, and at other times retention owing to the presence of blood clots in the urethra. Death eventually results from exhaustion.

Anatomical Changes.—In the early stages all that may be observed is red spots on the mucous membrane of the bladder or raised red ridges. In long-standing cases there is a fibrous thickening of the bladder with the formation of papillomatous growths which may be described as red bleeding warts.

Treatment.—Treatment is not very hopeful. A complete change of food and generous feeding may be resorted to.

Congestion of the Kidneys

Congestion of the kidneys may be *active* or *passive*. Active congestion is arterial hyperæmia; passive congestion is venous hyperæmia.

Active Congestion of the Kidneys.—This condition may be due to anything which dilates the renal arteries or increases the blood pressure in the aorta, thereby bringing about an increased flow of blood to the kidneys and resulting in an increased secretion of urine. Some drugs have this effect, such as potassium nitrate and other simple diuretics. It may also be produced by the presence of deleterious substances in the blood arising either in the course of various fevers, such as glanders and tuberculosis, or as a result of dietetic errors. Foods responsible for the condition include mouldy foods, foods containing large quantities of sugar, such as locust beans and mowburnt hay; fumigated oats may set up active congestion of the kidney owing to the presence of sulphurous acid. Active congestion of the kidneys may also be produced through the nervous system, as in the case of brain affections, interfering with the mechanism which regulates the flow of blood to the skin and the kidneys. It may also occur as the result of shock or fright, and injuries in the neighbourhood of the loins may also have the same effect.

Active hyperæmia of the kidneys as such is not a serious condition, but it may pass on to nephritis.

Symptoms.—The first thing observed is increase in the amount of urine passed; it is lighter in colour than normal and of reduced specific gravity. There may be some stiffness of gait, particularly of the hind limbs, which may cause rolling or staggering in progression, and in bad cases there may be some extra tenderness to pressure over the loins. The respiration is not interfered with, and unless it is due to bacterial origin there is rarely any alteration of temperature. In the majority of cases the condition passes away in two or three days if the cause is removed. If, however, the cause remains in operation it leads to considerable loss of condition and a staring harsh condition of the coat, and may pass on to nephritis. In rare cases of acute congestion there may be some hæmorrhage into the tubules of the kidney, resulting in hæmaturia.

Treatment.—First administer a purgative in order to relieve the functions of the kidney and to encourage excretion of the deleterious products by other channels. Apply rugs and bandages, and keep the body surface warm and so relieve the kidneys. A complete change of food and water should be obtained. Medicinally, nitrates and general diuretics should be avoided. Sulphate of magnesia in the food or drinking-water in small doses affords relief, and potassium iodide reduces thirst and diminishes the intake of water. A course of general tonics such as nux vomica and gentian may be administered.

Venous Congestion of the Kidneys ; Engorgement of the Kidneys.—In this condition there is an increased amount of blood present in the renal vessels, but a diminution of the amount of blood actually flowing through the kidneys,—a condition of partial stasis. Venous congestion of the kidney may be caused by any obstructions to the outflow of blood from the kidney, such as pressure on the renal veins as from a tumour or enlarged mesenteric gland, thrombosis of the renal veins or the posterior vena cava, valvular disease of the heart, particularly of the right side, and destructive lung disease raising blood pressure on the right side of the heart. This condition is much more serious than arterial congestion.

Symptoms.—There is first an increase in the amount of urine passed, which is followed in a day or two by a decrease in the amount owing to the reduction of the blood current. If the obstruction to the flow of blood to the kidney is very pronounced the urine would be markedly curtailed and a varying degree of albumen may be present. There may also be some hæmaturia. There is considerable loss of condition and frequently some degree of constipation. The pulse varies according to the cause, but is usually soft and large and lacking in tone. There may be some unusual excessive flinching on pressure over the loins. The gait is interfered with—a staggering with the hind limbs and debility of progression. The skin becomes shiny and glossy, owing to its increased activity on assuming some of the functions of the kidneys. If the condition lasts long the skin may become somewhat irritated by the abnormal excretions which should have been eliminated by the kidneys, and may become scaly, and sometimes emits a urinous odour. This condition is called urinous eczema. The primary cause of the venous congestion of the kidney may also be discovered, such as the heart affection, which is generally also associated with dropsy and venous congestion of the liver. When both kidneys are severely affected there is a danger of uræmic poisoning with convulsions or great depression followed by coma and death.

Anatomical Changes.—On post-mortem examination the kidney is found to be enlarged and is a dark, livid colour. On section the kidney substance is darker than normal and the Malpighian bodies may be prominently observed. In very old-standing cases the kidney becomes indurated and reduced in size, a condition which has been called *cyanotic induration of the kidney.*

Treatment.—Place the patient under comfortable conditions in a warm place and put on rugs and bandages. Immediate relief may be afforded by venesection or by the administration of a saline purgative. Aloes should be avoided, since it so frequently acts as a diuretic, which is contra-indicated in chronic venous congestion. For a similar reason one should avoid potassium nitrate and nitrous ether. Attention must also be directed to the primary disease, the best results being obtained by a combination of digitalis, nux vomica, and potassium iodide. Digitalis must, however, be used with caution in obstructive kidney disease, as it may become cumulative and poisonous. If a stimulant is required ammonia is the best for the purpose.

Nephritis ; Inflammation of the Kidneys

Pathologically, several forms of nephritis are recognized, viz. :

1. Parenchymatous nephritis, which includes (*a*) Tubular nephritis and (*b*) Glomerulo-nephritis.
2. Interstitial nephritis.
3. Suppurative nephritis.

Tubular nephritis is that form in which the renal epithelium is mainly involved. **Glomerulo-nephritis** is that variety in which the Malpighian bodies are mainly involved. **Interstitial nephritis** is that form which mainly affects the inter-tubular connective tissue. **Suppurative nephritis** includes all those cases in which pus is formed in the kidney.

Causes of Nephritis.—Nephritis is always the result of some irritation of the kidney substance. The injurious or irritating substances may be chemical, as in poisoning by cantharides or turpentine, or they may be deleterious substances formed in the body or absorbed from the alimentary tract and excreted by the kidney. These include bacterial products produced in other parts of the body in acute infections, as the result of which the kidneys may suffer even though the bacteria themselves may not be present in the kidney. Bacteria present in the kidney are a frequent cause of inflammation, and this is particularly the case in suppurative nephritis. Organisms may invade the kidney by way of the blood stream in septicæmic affections, in which case both kidneys are likely to be involved, or from the bladder by way of the ureter, in which case it is common to find one kidney alone affected. In females pregnancy is stated to be not infrequently an unsuspected cause of nephritis. Exposure to cold is still considered by many to be a cause of nephritis; cold undoubtedly may bring about a temporary congestion of the kidneys, and in this way may reduce the vital activity of the tissues and become a predisposing cause of nephritis. It is doubtful if cold by itself is ever an exciting cause of inflammation of the kidneys. Injuries in the region of the loins, such as contusions and falls, are said to result in nephritis, but the actual *modus operandi* is difficult to explain.

Clinically, nephritis may be considered under three headings, namely:

Acute nephritis, *chronic* nephritis, and *suppurative* nephritis.

Acute Nephritis.—This condition is in the majority of cases a parenchymatous nephritis involving the tubules. Acute interstitial nephritis may also occur. The difference between the two is very difficult to diagnose during life, but assistance may be afforded by microscopic examination of the urine. In the former case epithelial cells will be present in considerable quantity, whereas in the acute interstitial nephritis, although there may be albumen there is little or no epithelium present.

Symptoms.—In the early stages of acute nephritis there is marked increase of urine passed which is light in colour and of low specific gravity, and if the affection is very mild there may be no other symptoms observed unless the urine be examined, in which case albumen may be present in small quantity. As the condition advances the urine becomes turbid and reduced in quantity, and this is particularly marked if both kidneys are affected. The specific gravity is increased and the urine contains considerable quantities of albumen, which may be in the form of the various casts, epithelial, hyaline and blood casts, and in very old cases of tubal nephritis fat casts. By this time there is arching of the back, stiffness of the hind limbs amounting to weakness, often with a rolling gait. There is pronounced tenderness on manipulation of the loins in the larger animals, whereas in the smaller animals the kidneys themselves can be palpated through the abdominal wall producing evidence of pain and discomfort. There are usually frequent attempts at micturition, which become less effectual as the disease advances. The appetite is capricious and in ruminants rumination may cease. The pulse in the early stages is small, but in the later stages becomes full and rather hard. The temperature varies considerably, but in acute nephritis it is generally raised from 2° to 4° F. The respiration may be somewhat blowing and hurried, but not exaggeratedly so. The coat becomes glossy and sometimes emits an odour of urine. In the later stages œdema takes place into the dependent parts, as under the breast and abdomen and the limbs. By this time the loss of condition has become very marked, and in fact in a few days advanced emaciation may be produced in all species, but perhaps most noticeably in the dog. The urine should be examined from time to time, in order to note the progress of the condition. A decrease in the amount of albumen present is to be regarded as a favourable sign, but it must be associated with increased brightness. If the diminution of albumen is associated with greater depression or a tendency to coma it is due to the tubules becoming choked and the condition becoming worse. In dogs and other animals capable of emesis, vomiting is a frequent symptom of nephritis. The bowels become somewhat irregular, with a tendency to constipation. If both kidneys are affected uræmic poisoning may be set up and is usually fatal.

The onset *of uræmia in its acute form* is marked by the appearance of general convulsions and increased debility with temperature at first high but soon becoming subnormal. This is followed by coma, collapse and death. In the more *chronic cases of uræmia* debility becomes progressive, and there is greater interference with the digestive tract, producing indigestion and constipation, followed by diarrhœa, convulsions, vomiting in carnivora, urinous odour from the body, coma, and death.

Prognosis.—Recovery from mild cases of nephritis may occur, but complete recovery in well-marked cases is comparatively rare. More frequently it results in the onset of a chronic albuminuria owing to the permanent alterations in the renal epithelium. In other cases it may lead to chronic interstitial nephritis. Animals in such cases remain permanently in an unthrifty condition, and are unable to perform any prolonged exertion. In other cases, uræmia is set up and death follows.

Anatomical Changes.—On post-mortem examination the kidneys are found to be larger and heavier than normal. On section the capsule is easily stripped off, and the cortex appears to be swollen, and there may be small hæmorrhages into it. In bad cases the catarrhal exudate may be observed in the tubules and the pelvis of the kidney.

Treatment of Acute Nephritis.—Place the patient under the best hygienic conditions, particularly with regard to warmth. Clothing and bandages should be applied, and hot blankets across the loins afford relief. Counter-irritants to the loins such as blisters of cantharides of turpentine must be *avoided.* Attention must be paid to the food, which should not be too highly nitrogenous. In herbivora the food should consist mainly of linseed mashes, linseed tea, bran mashes, barley water, milk, and hay tea with a little steamed hay of first quality. For the carnivora a milk diet is best associated with concentrated foods and infant foods such as corn-flour, Benger's food, virol, and meat essences. Cod-liver oil and malt are also very useful. Medicinally, a purgative should first be administered, preferably either a saline or calomel. This may be followed by digitalis with acetate of ammonia, and belladonna or hyoscyamus. Renal antiseptics may also afford relief, such as hexamine, salicylates, and the balsams. Stimulating diuretics such as potassium nitrate and spirits of nitrous ether should be *avoided.* If there is any danger of uræmic poisoning venesection should be performed, and this should be followed by potassium bromide or chloral hydrate. In these cases, however, treatment is not likely to have any beneficial results.

Chronic Nephritis.—This may be a chronic parenchymatous nephritis or chronic interstitial nephritis. In each case both kidneys are usually affected, and the symptoms during life are practically indistinguishable. They may come on as the sequel to acute nephritis, although occasionally they may be primary in origin.

Symptoms.—In chronic nephritis there may be the history of a previous attack of acute nephritis, from which partial recovery has occurred. When no such history is available the first symptoms are those of general unthriftiness, which develops slowly and gradually in spite of good appetite and generous diet. The patient becomes fatigued on slight exertion; urine is discharged frequently and in large quantities and is albuminous. The amount of albumen varies. When it is copious the wasting and loss of flesh are very rapid, and the urine churns into a froth as it falls on the ground, the froth persisting a considerable time. There is a tendency to œdema of the dependent parts such as the limbs and under the breast and scrotum, and also dropsy of the cavities, particularly ascites. The back is arched, and the loins tender to manipulation, but less marked than in acute nephritis. There is great debility with staggering gait. The appetite becomes capricious; there is considerable thirst for water and frequent vomiting in animals capable of emesis. The temperature is normal or slightly subnormal, the pulse usually large and hard. As the condition progresses the functions of the kidney become reduced, and there may be suppression from one or both kidneys. If both kidneys are thus affected it rapidly proves fatal. If only one kidney is involved the remaining kidney becomes hypertrophied and takes on a double function. Cutaneous complications may also be met with in the form of scaly eruptions associated with an offensive urinous odour. This condition is called *urinous eczema*, and is comparatively common in the dog, giving rise to considerable pruritus. The precise etiology of the so-called urinous eczema is, however, obscure.

Interstitial nephritis often leads to hypertrophy of the left ventricle of the heart in cases where the patient was previously in good muscular tone, in other cases it leads to dilatation of the ventricle.

Anatomical Changes.—In *chronic parenchymatous nephritis* both kidneys are equally affected. They are considerably enlarged and yellowish or dirty white in colour; the capsule is easily stripped off. When cut the increase of size is found to involve the cortex mainly, showing greyish-yellow or greyish-red areas. On scraping the surface fat droplets are demonstrated. *In chronic interstitial nephritis* one or both kidneys may be affected. They are generally reduced in size, maybe to one-third their normal volume. The capsule is very adherent and only stripped off with difficulty, the surface of the cortex being more or less pitted in appearance. On section the reduction in size has seemed to affect the cortex mainly, which is also tougher than normal. In some cases cysts varying up to the size of peas may be found in the cortex. The liquid contained is urine collected as the result of dilatation of tubule above a constriction. In those cases where one kidney alone is involved it is found to be very much reduced in size and tougher than normal, but the other kidney is larger than normal, having undergone hypertrophy.

Treatment.—Curative treatment of chronic nephritis is hopeless, but palliative treatment may be resorted to. A mild purgative should be administered, either saline or calomel, and the bowels should be kept in a more or less relaxed condition by a laxative diet. In the horse and ox the administration of linseed oil ℥ii.-℥iv. once or twice daily with the food is helpful. In dogs and cats paraffinum liquidum ʒi. or ʒii. may be given twice a day mixed with the food. Raw ox liver may also be given in these cases to assist in regulating the bowels. The extremities must be kept warm. Careful attention must be paid to the diet, and the food must be of unimpeachable quality and not too highly nitrogenous. Medicinally, repeated small doses of calomel for the horse and ox, and grey powder for dogs, are serviceable in maintaining intestinal excretion and so relieving the kidneys. A course of potassium iodide may also be administered to reduce the general dropsical condition of the patient and to allay thirst. This may be combined with small doses of nux vomica. In the smaller animals a combination of cod-liver oil, malt, and iron will be found very useful. Particular care must be exercised in the administration of poisons in obstructive kidney disease, as they are likely to become cumulative. This is especially the case with digitalis and belladonna. The cutaneous complications must be treated as they arise. In dogs and cats beneficial results often follow the hypodermic injection of 1 c.c. to 5 c.c. of a 1 per cent solution of collargol. Good grooming and careful attention to the condition and cleanliness of the skin are essential, and the pruritus may be relieved by the application of zinc ointment. In cases where uræmia is set up treatment is not hopeful.

Suppurative Nephritis ; Purulent Nephritis ; Pyonephrosis.—Suppurative nephritis is due to pyogenic organisms, which may be carried to the kidney by the blood stream or may gain access from the bladder by way of the ureters. When brought by the blood stream the condition is known as *pyœmic nephritis*. It is always secondary to suppuration in another part of the body, and both kidneys are usually affected.

When organisms gain access by way of the ureters and the pelvis of the kidney, the condition is called *pyelonephritis*. When the pelvis alone is affected it is called *pyelitis*, and this usually precedes pyelonephritis. In this form it is usual for one kidney alone to be affected.

Symptoms.—*Pyœmic nephritis* is often very difficult to diagnose, but one is usually assisted by the history of the previous attack of some purulent affection such as strangles in the horse. In some instances there appear to be no symptoms exhibited during life, and the condition is found on post mortem without the true nature having been observed. In the majority of cases, however, earlier symptoms correspond with those of acute nephritis, stiffness in progression, and staggering gait, with debility of the hind limbs and some pain across the loins, the temperature oscillating, as is frequently the case in pyogenic affections. In the earlier stages there may be no albumen or pus in the urine, but later, when the abscess has burst, both may be found in the urine on chemical and microscopic examination. In many cases, however, when the abscesses are very numerous, the condition proves fatal before they have time to attain any considerable size and before any pus is discharged in the urine. Positive diagnosis in such cases is impossible.

The symptoms of pyelonephritis are generally preceded by an attack of cystitis. There are frequent and painful attempts at micturition, and the patient often does not assume the ordinary position to stale. There is the usual interference with gait, and tenderness of the loins is more pronounced on one side than the other, since the condition is generally unilateral. The urine is albuminous, containing pelvic epithelium, pus cells, blood, and occasionally blood casts of the pelvis. Tubular casts are not often met with. The temperature is oscillating. This condition, although a serious one, is not so likely to prove fatal as pyæmic nephritis, owing to the fact that there is usually one kidney remaining normal to perform its functions.

In pyelitis the symptoms are scarcely distinguishable from those of pyelonephritis except in those cases where it is due to renal calculus. In that case there are periodical attacks of obscure colic associated with spasmodic urination, and the urine is blood-tinged for some time. In some cases the outlet from the kidney to the ureter is completely closed, urine then accumulates, the renal substances undergoing atrophy from pressure, and in this way the kidney may become converted into a large renal cyst, to which the name *hydronephrosis* is applied.

Anatomical Changes.—On post-mortem examination in *pyœmic nephritis* both kidneys are found to be more or less swollen, and a number of abscesses may be seen under the capsule and also on section. They may be very numerous and very small, and are generally met with in the cortex. When very small they might be mistaken for miliary tubercles. Between the abscesses there may be congestion and some hæmorrhages. In *pyelonephritis* one or both kidneys may be found affected. The kidney is considerably enlarged, and more or less large abscesses may be seen even before the kidney is cut. On section the abscesses are found principally in the medulla of the kidney, especially if seen at an early stage. In *pyelitis* the pelvis of the kidney contains a muco-purulent material sometimes blood-stained, and in old-standing cases is dilated.

Treatment.—In suppurative nephritis it is doubtful whether any beneficial results can be obtained by treatment. In the pyæmic variety the cases will no doubt prove fatal in a comparatively short time, but in pyelonephritis life may be prolonged by palliative measures. A course of urinary antiseptics should be administered, such as hexamine (to which should be added sodium acid phosphate in the case of herbivora, since it is more potent as an antiseptic in a slightly acid medium), acetosalicylic acid, salol, balsam of tolu, and benzoin. Belladonna and hyoscyamus are the best sedatives to use. The bowels should be kept relaxed and free with salines and with appropriate diet. Hot applications to the loins also afford relief. If the causal pyogenic organism can be recovered from the urine an autogenous vaccine may be tried, but it is doubtful if it is of much service.

In *pyelitis* the treatment is on exactly the same lines, and is rather more hopeful of being followed by beneficial results.

Renal Calculus ; Stone in the Kidney

Amongst the domesticated animals urinary calculi are not infrequently met with in horses and cattle, and more rarely in the smaller animals. They are probably more common than is generally realised, owing to the difficulty of diagnosis during life, and the kidneys not being systematically examined after death. This fact is shown by the frequency with which they are met with in horses at the knacker's when looked for, and in ox kidneys at the abattoir. They are more frequently met with in old than in young animals.

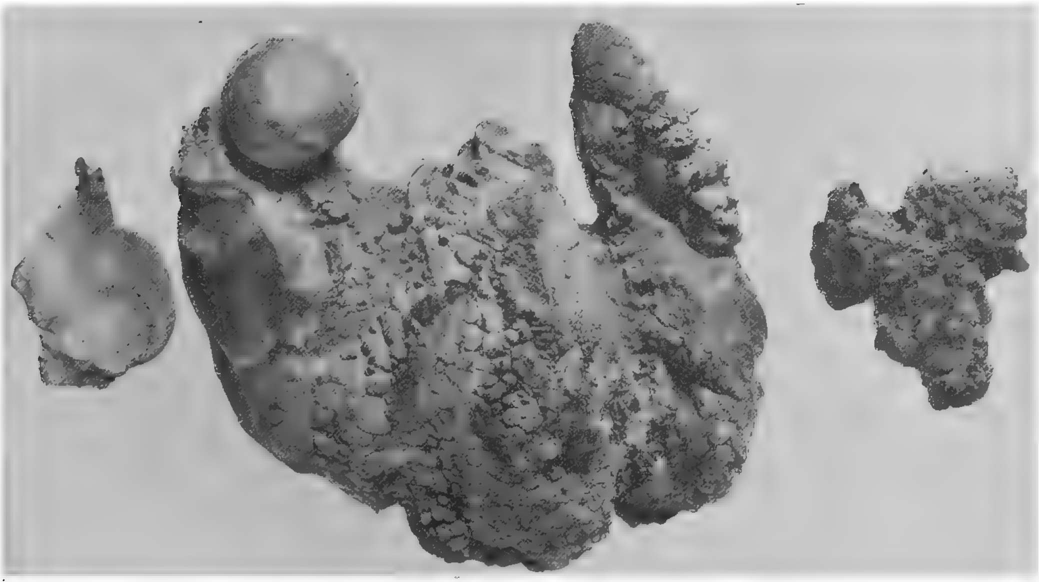


FIG. 75.—Calculi from the kidney of a horse. ($\frac{3}{5}$ actual size.)

Causes.—Renal calculi are caused by the slow and gradual deposition of salts from the urine, usually round a nucleus which may consist either of a small amount of epithelial cells, pus, shreds of fibrin, and sometimes bacteria. They may therefore follow nephritis due to any cause. The method of feeding is probably an important factor, foods rich in phosphates, such as bran, and food-stuffs rich in calcium salts being blamed for the condition. Attempts to produce renal calculi by ordinary food-stuffs have, however, been negative, although in experimental cases renal calculi have been observed by Thomassen in a dog which had been fed with oxamide. Similar results have also been observed by Ebstein and Nicolaier (quoted by Hutyra and Marek).

Composition and Form.—Renal calculi in herbivora are mainly composed of carbonates of calcium and magnesium, together with calcium oxalate and organic matter. **In the horse** they vary in size and may attain a weight of several pounds. They are generally very rough and irregular in outline, due to being moulded in the pelvis and calyces of the kidney. The largest have a crescentic convex outline which corresponds with the pelvis of the kidney, and along the other margin they carry projections which protrude into the substance of the kidney. The renal tissue has undergone considerable atrophy and in some instances almost entirely disappeared. The calculi are occasionally multiple, greyish white in colour, and they have a high specific gravity. In **cattle and sheep** renal calculi are usually multiple, have a smoother surface, and are yellowish grey or yellowish brown in colour. They are not so heavy or dense as those of the horse. In **carnivora** renal calculi are usually small and very irregular in outline, greyish yellow in colour, and consisting largely of phosphates of calcium, magnesium, and ammonium, together with calcium carbonate and some urates.

Symptoms.—There is frequently difficulty in diagnosing renal calculus, particularly in the early stages. The earlier symptoms are those of nephritis and pyelitis, with some loss of condition, arched back, straddling gait, and unthriftiness. The urine is albuminous, containing epithelium and serum albumen, and is frequently blood-stained, while the latter portion of urine passed may contain a sandy deposit, which can be seen on the ground immediately after micturition, thus differentiating it from so-called oxaluria in horses due to indigestion, in which condition a sediment is frequently produced some time after the urine has been passed. In many cases the patient becomes affected with intense spasmodic pain, the spasms lasting a considerable time, maybe an hour or more, with frequent attempts at micturition, and vomiting in animals capable of emesis. The attacks of so-called renal colic occur quite irregularly and there may be intervals of months. They are probably due in many cases to the passage of a small calculus down the ureter into the bladder. Examination per rectum in the larger animals, and palpation through the abdominal wall in the smaller animals, may yield grounds for a positive diagnosis. As a general rule, only one kidney is involved; the remaining kidney takes up the double function, and consequently there is rarely or never uræmic poisoning.

Treatment.—It is impossible to cause any re-solution of the salts in the kidney when once calculi have been produced. One's efforts must therefore be directed to palliation. Attention must be paid to the diet, and foods containing excessive quantities of salts, such as bran, should be avoided for herbivora, and also beans and peas and immature mangolds, boiled linseed being substituted for them. In carnivora the meat rations should be reduced, bones being avoided and barley water being given to drink. In each case soft water should be supplied. Urinary sedatives and antiseptics must be administered, such as belladonna or hyoscyamus and hexamine. Attempts should be made to flush out the kidneys with urine as nearly neutral in reaction as possible. For this purpose nitro-hydrochloric acid and salicylates and sodium acid phosphate may be given to herbivora to reduce the normal alkaline reaction of their urine, and alkalies such as bicarbonates of sodium or potassium to carnivora for the opposite reason. Lithium salts are also frequently recommended. In cases of renal colic, morphia or chloral hydrate may be administered, hot cloths applied to the loins, and hot enemata administered. In the smaller animals surgical interference may be necessary, and when unilateral the affected kidney may be removed.

Parasites of the Kidneys

Renal parasites are comparatively rare, but the following are sometimes met with:

Eustrongylus gigas in dogs, horses, and cattle.
Larvæ of *Strongylus vulgaris* in horses.
Stephanurus dentatus in pigs. (See "Parasites of Peritoneum," p. 339.)
Cystic stages of tapeworms (*Cysticercus cellulosæ* in pigs and chinococcus cysts in ruminants). And more rarely *coccidia* are said to have been observed in the kidneys of horses and dogs and also in geese.

Eustrongylus gigas.—This parasite is met with in the pelvis of the kidney in dogs, horses, and cattle in S. America or animals imported from there. It varies in length from 6 to 18 inches in the male to 2 or 3 feet in the female, and has the thickness of a goose quill. It is reddish in colour. The parasite is usually found curled up in the pelvis of the kidney surrounded with blood-stained purulent material resulting from the inflammation it sets up there. There is usually only one parasite present, but occasionally more are found.

Symptoms.—These vary very considerably, and sometimes the presence of worms has not been suspected until they are discovered on postmortem examination. That is particularly the case when occurring in horses and cattle. In dogs, however, symptoms are more frequently observed. They consist of those of pyelitis with general loss of condition and emaciation, and sometimes with frenzy. The urine is albuminous and may contain pus and blood. Positive diagnosis, however, can only be made in the majority of cases by the discovery of the eggs of the parasite in the urine. Occasionally the parasite is said to leave a kidney by way of the ureter and to be expelled during micturition in the larger animals, while in the dog it becomes arrested in the urethra, causing retention of urine and a considerable swelling in the perineum the size of an orange in which the worm is coiled up.

Treatment.—Attempts may be made to remove the parasite by the administration of turpentine by the mouth. If that fails, as it is very likely to do, surgical measures (nephrotomy or urethrotomy according to circumstances) may be taken in the case of the smaller animals.

Cystitis; Inflammation of the Bladder

The causes of cystitis are irritants in the bladder which gain access either by way of the kidneys or by way of the urethra. They may thus consist of irritants in solution of the urine, as cantharides and turpentine, or they may be bacteria, which are undoubtedly the most common cause. The organisms may have passed into the bladder from the kidneys in nephritis or they may have invaded the bladder from the urethra in urethritis, or metritis or vaginitis in females, or prostatitis in males. Organisms may also be introduced by means of dirty catheters. Many bacteria which are not usually regarded as pathogenic may set up cystitis when introduced into the bladder by causing decomposition of the urine and producing substances very irritant to the mucous membrane. The presence of a calculus in the bladder or sabulous material (sand-like deposit or "gravel") may also set up cystitis. This sandy material is comparatively common in ruminants, and especially in rams fed on mangolds in the early part of the winter. Anything which causes retention of urine predisposes to the deposition of salts, and also favours the development of bacteria when once they have gained access. This is particularly observed in castrated cats owing to their sedentary habits.

Pathologically various types of cystitis may be met with, namely, catarrhal, croupous, diphtheritic, purulent, and hæmorrhagic cystitis. *Clinically*, however, there is no necessity for this differentiation, but *acute* and *chronic cystitis* are recognized.

Acute Cystitis; Symptoms.—Usually the first indication of cystitis is frequent attempts at micturition and evidence of pain during the process. Urine is passed spasmodically and in small drops or jets (strangury). In some cases where the neck of the bladder is involved there may be incontinence or dribbling with excoriation of the perineum in the female, while in the gelding or stallion the penis may become pendulous and swollen and set up paraphimosis. In other cases spasm of the neck of the bladder occurs affecting the sphincter, and in these cases the desire to micturate is increased but the ability is lost, and consequently great pain and colic are produced. On examination of the bladder per rectum in the larger animals, assisted by abdominal palpation in the smaller animals, pain is evinced when there is any pressure applied to the bladder. The temperature is elevated if the condition is bacterial in origin, but not when dietetic or due to calculi. The pulse is accelerated, and in painful cases the mucous membranes are injected. There is frequently constipation, largely voluntary owing to the pain produced by pressure on the bladder during defæcation. In dogs and cats vomiting also frequently occurs. On examination of the urine it is found to be albuminous with increased specific gravity, and may be blood-stained and frequently offensive in odour. It may also contain a considerable amount of mucus, especially in the horse, causing an oily consistence which is described as being "ropy" or "stringy." When the cystitis is deep-seated the urine also frequently contains flakes, which may be due to coagulated croupous exudate or to necrosis of the superficial layers of the mucous membrane. On microscopic examination of the urine, bladder epithelium may be recognized. The urine is usually rich in organisms of various kinds, the colon type being frequently present in profusion. At other times very small mulberry-shaped calculi may be seen. When there is much hæmorrhage into the bladder in cystitis the urine may be uniformly blood-stained, and irregular clots of blood will be expelled with the last portion of the urine passed. Cystitis is very common in castrated male cats and is associated with catarrh of the urethra and the deposition of salts, almost invariably causing retention of urine, vomiting, constipation, semi-coma, and collapse.

Treatment.—Absolute rest is essential to successful results. If urine is retained it must be removed by the catheter, strict attention being paid to the cleanliness of the instrument. In some cases in the smaller animals it may be necessary to puncture the bladder with a small trocar and cannula, although frequently pressure exerted through the abdominal wall *with care* may empty the organ. Unless care is used the bladder may be ruptured. The bladder should be irrigated through the catheter in the larger animals with a warm non-irritant antiseptic, such as eusol, saturated solution of boracic acid, chinosol (1-2000), corrosive sublimate (1-5000), or carbolic acid or salicylic acid from ½ to 1 per cent. A simple purgative should be administered, such as oil, or aloes in the larger animals or calomel in the dog and cat. A course of urinary sedatives and antiseptics should be prescribed. Where the condition is very painful, hyoscyamus or belladonna afford considerable relief, but they must be used with care if there is any hæmorrhage. In such cases tincture of buchu and oil of sandal-wood are better. Probably the best antiseptic for the bladder is hexamine, which is more effective when the urine is rendered acid in reaction. It should therefore be combined with sodium acid phosphate or sodium salicylate. Hot applications across the loins and fomentations in the perineum afford relief, as also may the injection of copious hot enemata. The application of a blister to the loins is contra-indicated. Rugs and bandages must be applied to maintain bodily warmth. Plentiful supplies of water should be prescribed and the drinking of water encouraged. The diet should be somewhat restricted and not of a stimulating character. In the larger animals, beans and peas should be avoided and mucilaginous foods given, such as boiled linseed and steamed hay and hay tea. In the smaller animals, boiled sheeps' paunches are very good, with milk and egg, and barley water to drink.

Chronic Cystitis.—This condition is due to similar irritants as acute cystitis operating in a less exaggerated degree. It may occur as a sequel to the acute form, and is also present in cases of calculus of the bladder.

Symptoms.—The symptoms of chronic cystitis are not very pronounced in the majority of cases. There is frequent micturition and often incontinence. The urine is turbid and may be blood-stained, and also contains bladder epithelium and sometimes pus cells. The condition is much less severe than in the acute form. In male cats chronic cystitis frequently leads to retention of urine of offensive odour.

Treatment.—This should be on the same lines as for acute cystitis. The bladder should be irrigated in a similar manner, and internally hexamine should be given combined with sodium acid phosphate and buchu or copaiva. In painful cases belladonna or hyoscyamus should be administered, but these should be used with caution if there is hæmaturia. In male cats when retention occurs it may be necessary to puncture the bladder daily for several successive days, and the perineum should be fomented. In many cases of this character in the writer's experience complete recovery has occurred after frequently repeated puncture.

Cystic Calculus ; Vesical Calculus ; Stone in the Bladder

Calculi occur in the bladder as the result of deposition of salts due to various causes, such as the presence of bacteria or catarrhal cells in cystitis. Retention of urine favours the deposition of salts, as also does concentration of urine in cases where there has been an insufficient supply of drinking water. The method of feeding is also an important factor, and this is particularly the case in rams fed on mangolds too early in the season. (Mangolds should never be employed for feeding cattle or sheep until they have been stored several months, *i.e.* until January or February.) As in the case of renal calculi, bladder calculi are formed round a nucleus which may consist of bacteria, epithelial cells, inflammatory exudate as shreds of lymph. Occasionally foreign bodies have been found in the centre of cystic calculi, such as bits of straw or metal, and sometimes bits from the broken end of a catheter. In some cases the calculi have been formed in the kidney, having passed down the ureter to the bladder, there to constitute a nucleus for the further deposition of salts and the growth of a larger cystic calculus.

Cystic calculi vary in size ; they may be very small and very numerous, appearing almost like sand or " gravel," or they may be as large as a goose's egg. The common shape of the larger ones is spheroidal or ovoid, somewhat flattened from above to below. The outer surface is generally regular or symmetrical, but roughened as though built up by the adhesion of large numbers of very small calculi. They also usually show one or two smooth areas indicating the points of attachment to the bladder wall. In swine when a single calculus is present its surface is usually much smoother than in other animals. In dogs a single calculus may be met with, or there may be several showing facets or flat surfaces where they have rubbed together, thus rendering them more or less pyramidal in shape. The calculi are usually very heavy and hard and dense. In the case of the dog, however, one frequently meets with them quite soft and pultaceous on the outside. The colour of cystic calculi is usually yellowish brown in horses, cattle, and sheep, in swine a greyish white, and in the dog both yellowish brown and white calculi are met with in the different cases.

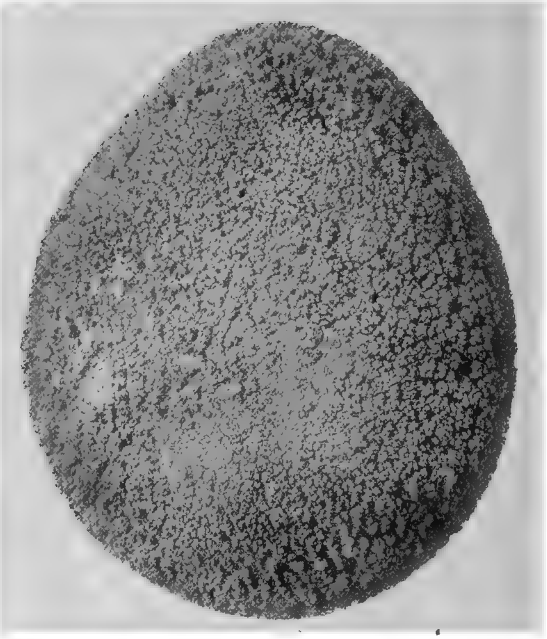


Fig. 76.—Calculus from the bladder of a mare. (¾ actual size.)

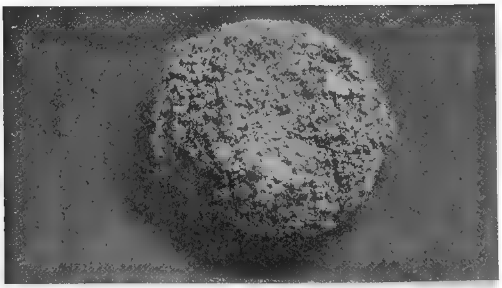


Fig. 77.—Calculus from bladder of Pekingese dog. (Natural size.)

Composition.—In the horse the majority consist of calcium carbonate together with traces of triple phosphates and some urates and oxalates. The lighter smooth calculi of other species such as the dog mainly consist of triple phosphates, but contain some carbonate and urates.

Symptoms.—Cystic calculi rarely cause trouble in the early stages of their development. After a time they give rise to frequent staling, and sometimes in the course of micturition there is a sign of sudden pain and a stoppage of the flow of urine, repeated attempts to stale being in vain. In the case of the horse and ox there is twitching of the tail and kicking forward at the belly. In other cases there is a dribbling of urine with excoriation of the perineum and thighs in the female and of the sheath in the male. In the horse (gelding or stallion) the penis hangs free from the sheath (paraphimosis). The urine is very turbid and frequently offensive in odour and smells of ammonia. There is also frequently intermittent hæmaturia. In such cases cystitis is also present, the symptoms of which may also be observed. Examination per rectum may reveal the presence of calculus, and assistance is also frequently obtained by the passage of the sound in the case of the female.

Treatment.—In the case of sabulous material in the bladder, irrigation with sterile saline solution or saturated solution of boric acid should be carried out. A plentiful supply of good water should be provided and the drinking of water encouraged. A smart purgative should be administered to relieve the kidneys of the excretion of salts, and a course of sedatives to the urinary tract administered, such as recommended in cystitis. The diet should be laxative in character. In the case of the large calculi, surgical measures must be resorted to (see Surgical Section).

Retention of Urine

Retention of urine may be due to stricture of the neck of the bladder, to paralysis of the bladder walls, to calculi lodged in the neck of the bladder or in the urethra, to malignant growths, and also to enlarged prostate pressing on the urethra. In the gelding it may be due to a collection of smegma in the coronal fossa in the glans penis. Spasm of the neck of the bladder may occur in some cases of colic and cause retention. Retention of the urine may also occur in some cases of spinal injury.

Symptoms.—In the first place there is a cessation in the passage of urine which gives rise to considerable pain; the patient frequently places himself in a position to micturate, but fails to do so, but in some instances there may be an escape of one or two drops of urine. In the larger animals acute spasmodic colic may be set up, and the distended bladder can be felt on examination per rectum. In the smaller animals the distended bladder can be felt through the abdominal wall, frequently very forward in the abdomen. In old-standing cases in the dog, and especially in the cat, there is repeated vomiting, urinous odour from the skin, and partial collapse. Retention of urine is very frequently complicated by constipation.

Treatment.—An effort must first be made to expel the urine by pressure on the bladder in the case of the larger animals per rectum, and in the case of the smaller animals through the abdominal wall. Great care must be taken, however, especially in long-standing cases, for fear of causing rupture. If this fails, the urine must be removed by the catheter in the larger animals, and in the smaller animals either by the catheter or by puncturing with a trocar and cannula through the abdominal wall. In the cat, care must be taken not to rupture the bladder during the process. The writer has also successfully resorted to prepubic puncture in the case of a calf three months old. Hot applications to the loins and the perineum will afford relief in painful cases. A smart purgative should be administered and the water supply curtailed. Attempts should then be made to remove the cause; if it is due to paralysis of the bladder or defective nervous or muscular force, a course of strychnine or nux vomica should be prescribed. If due to enlarged prostate it should either be removed surgically, or it may be reduced by a course of potassium iodide and potassium bromide. Castration also assists in the reduction of enlarged prostate. If the retention is due to calculus or malignant growth the treatment is purely surgical.

Incontinence of Urine

This is inability to retain urine in the bladder. It is often brought about by injury to the bladder wall, particularly in the region of the neck, or to paralysis of the sphincter of the bladder, and also to calculus or tumours. It may be due to nervous causes and fright, especially in very young and very old animals. Incontinence may also follow injury to the spine in the region of the loins.

Symptoms.—There is more or less constant dribbling of urine, causing excoriation of the perineum and thighs of the females and of the sheath or prepuce in males. There is a urinous odour as the result of the dribbling, and in the majority of cases loss of condition and general debility.

Treatment.—Where the cause can be found treatment must be directed towards it. Calculi or tumours should be removed. If due to cystitis, corresponding measures should be adopted. If due to nervous causes, a purgative should first be given, and this should be followed by a course of strychnine or arsenic or a combination of the two.

Paralysis of the Bladder

This condition may be due to nervous causes. Not infrequently it is due to the enforced retention of urine causing over-distension and muscular paralysis. It may lead to retention of the urine, while in other cases, if the neck of the bladder is involved, incontinence of urine is produced.

The *symptoms* will depend therefore upon the primary cause of the condition, and are those either of retention or incontinence.

Treatment.—This depends upon the cause and upon the symptoms produced. If necessary the catheter must be passed frequently, but great care and all antiseptic precautions must be taken owing to the danger of producing cystitis. The application of a blister of biniodide of mercury across the loins may assist in restoring the nervous force. The application of the galvanic battery may be resorted to, but its efficacy is open to doubt. A course of nerve tonics should be administered such as strychnine and arsenic.

Hæmorrhage in the Bladder

This condition is also referred to under "hæmaturia" (p. 341). It may be due to calculus, to cystitis, or to acute irritant poisons such as cantharides. It may also be due to tumours of various character in the bladder. Wooldridge has met with several cases in the dog due to hæmorrhagic papilloma.

Symptoms.—The passage of urine which is generally diffusely blood-stained and may also be followed with irregular blood-clots with the latter part of urine discharged. In the horse the amount of blood is sometimes very excessive, and one occasionally meets with cases where the urine appears almost as if it were pure blood.

Treatment.—The cause should be discovered wherever possible and treated accordingly, calculi and tumours removed, cystitis treated. The hæmorrhage may also be checked by irrigating the bladder with a weak solution of adrenalin, 1-10,000, or extract of hamamelis or alum 2 per cent sol. Internally tincture of buchu may be given, but belladonna and hyoscyamus are contra-indicated since they relax the blood-vessels and tend to increase the hæmorrhage.

Urethritis ; Inflammation or Catarrh of the Urethra

Urethritis is due to an irritant in the urethra. It may be infective or contagious and spread during coitus. In the female it may be secondary to metritis or to vaginitis. In either sex, urethritis may precede or be secondary to cystitis. Urethritis may also be brought about by calculi arrested in the urethra, by injury in the use of the catheter, or injury to the free extremity of the urethra or in the region of the perineum in the male.

Symptoms.—The first thing observed is frequent and painful micturition, and between the acts of urination drops of catarrhal exudate may be seen. In mild cases, in the male, erection of the penis is frequently produced, and some nymphomania or sexual excitement in either sex. In the female the irritation frequently extends to the vagina and vulva, and in the male to the sheath, but one should differentiate the latter condition in the dog from catarrh of the sheath, which is extremely common and is due to dribbling of the last few drops of urine into the sheath. In the gelding and stallion the penis frequently becomes pendulous and swollen, paraphimosis resulting. In long-standing cases the urethra may become considerably thickened, causing retention of the urine, and in other cases leading to permanent stricture of the urethra. In male cats it is common to find a small bit of sand-like material in the free portion of the urethra which can be removed by careful manipulation with the assistance of a fine bougie and a little oil.

Treatment of Urethritis.—The urethra should be injected with a non-irritant antiseptic and astringent. Useful preparations consist of saturated solution of boracic acid, chinosol 1-1000 to 2000, or carbolic acid 1 per cent solution. Weak astringents may also be employed, such as sulphate of zinc grs. iv.-v. to ℥i. or sulpho-carbolate of zinc or acetate of lead in similar strengths. Another very useful preparation is collargol from 1 per cent to 5 per cent solutions. One must be careful not to use too potent astringents as they may contribute to the production of stricture. Internally sedatives and antiseptics to the urinary tracts should be administered, such as hexamine and copaiva and oil of sandal-wood. Belladonna may also be found useful in many cases, while potassium bromide may be administered to reduce erections and sexual excitement. An effort should be made to render the urine neutral in reaction, giving sodium acid phosphate to herbivora, and bicarbonate of soda or potash to carnivora. The drinking of water should be encouraged to dilute the urine and to reduce the painful effects of a concentrated urine passing over an inflamed mucous membrane.

Stricture of the Urethra

This is a surgical condition which may result from urethritis or injury to the urethra from other causes, such as the faulty passage of the catheter, urethral calculus and operation for the same, and also external injuries, especially in the male, in the region of the perineum. The writer has also observed constriction of the urethra in the dog as a result of exostosis involving the furrow of the os penis. Stricture may also occur at the free extremity of the urethra as the result of tumours, and also after surgical amputation of a portion of the penis. Although it may occur in both sexes, it is most frequently met with in the male.

Symptoms.—The most important feature is difficulty of micturition. Urine is passed in a very small stream, or may be retained accompanied by considerable pain and the general symptoms of retention of urine. Diagnosis is confirmed by the vain attempt to pass the catheter.

Treatment.—It may be necessary to relieve the bladder first by the trocar and cannula. This, however, is only palliative; attempts must be made to dilate the stricture by means of catheters or bougies, commencing with those of small calibre and gradually increasing in size. The instrument should be first well lubricated, and when inserted should be allowed to remain in position for ten or fifteen minutes. It may be necessary to repeat the operation twice daily. The application of a small amount of extract of belladonna to the point of the instrument may assist in the operation by somewhat relaxing the urethra.

Urethral Calculus

Urethral calculi generally consist of small vesical calculi that have been passed from the bladder and become arrested in the urethra. They are rarely met with in the female; they are more common in the male, owing to the greater length and the course of the urethra. They are somewhat rare in the horse, rather more common in the bull, and still more frequently met with in the ram and in the dog. Their position in the horse may be at the ischial arch or at the point of the penis. In the bull they may be found at the ischial arch or at the S curve of the penis. In the ram the common seat is at the free extremity of the penis, where they become arrested at the prolongation of the urethra beyond the glans penis (*i.e.* at the commencement of the so-called vermiform appendix). In the dog urethral calculi may be met with at the ischial arch, but are more frequently lodged in the groove of the os penis. In the cat similar obstructions of the penis, particularly the castrated male, are due to very tiny calculi or to sabulous material arrested at the free extremity of the penis. In the horse similar symptoms may be produced by smegma in the coronal fossa at the point of the penis.

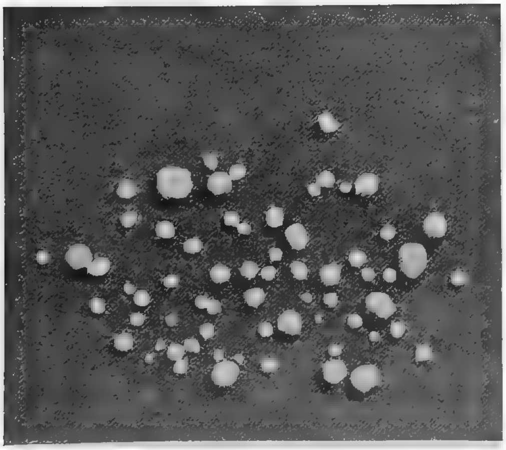


FIG. 78.—Urethral calculi from a small terrier. (Natural size.)

Symptoms.—Urethral calculi may cause complete or partial retention of urine together with urethritis. There are frequent attempts at micturition with only a few drops of blood-stained urine passed. There may also be the oozing of a drop of urine or of blood from the point of the penis between the acts of micturition. If the calculus is at the ischial arch it may be felt on manipulation of that region; if, however, the obstruction is lower down the urethra the structure behind the obstruction becomes distended with urine and considerable pulsation or throbbing may be felt on manipulating it. The bladder may also be found distended.

Treatment.—The treatment of urethral calculi is usually surgical, although in the case of the cat careful pressure on the bladder through the abdominal wall, together with manipulation of the penis with the thumb and finger, may serve to dislodge the obstruction. (For treatment in other animals see Surgical Section.)

Parasites of the Urethra

As already mentioned, the eustrongylus gigas may be met with in the urethra, being arrested on its way out from the kidney.

Prostatitis ; Inflammation of the Prostate

This condition may be met with in uncastrated males of all species of animals.

Causes.—Prostatitis is generally due to micro-organisms. Infection may take place by ascent of the urethra or may be secondary to cystitis; or bacteria may be carried by the blood-stream from other parts of the body as in strangles in the horse. Prostatitis may also result from repeated attacks of retention of urine and from injury to the duct of the prostate during the passage of a catheter. In dogs it may be secondary to impaction of the rectum.

Clinically prostatitis may be *acute* or *chronic*. The chronic form may be preceded by an acute attack, but in a large proportion of cases, particularly in dogs, it is primary and comes on insidiously.

Acute Prostatitis.—*Symptoms.*—In mild cases there are only few symptoms. In the early stages frequent micturition and sometimes incontinence of urine. In more severe cases there is painful swelling of the perineum and retention of urine. On passing the catheter pain is evinced when the instrument reaches the prostate. Examination per rectum reveals the enlarged gland, which is very painful to manipulation. The temperature is raised from 2° to 4° F. There is a rolling gait and weakness of the hind limbs, which in dogs may amount to partial paraplegia. Abscess formation may take place, causing considerable distress. When mature the abscess may burst either into the urethra, into the perineum, or into the rectum. After the abscess has burst the symptoms are somewhat allayed. The condition is frequently mistaken for cystitis, but in that condition the urine is usually turbid throughout, while in prostatitis the bulk of urine is clear up to the latter portion, which contains prostatic fluid. This may be detected microscopically by the precipitation of long-pointed crystals in the fresh preparation to which has been added a little ammonium phosphate solution.

Treatment.—Absolute rest is essential. Hot fomentations should be applied to the perineum and hot enemata administered. These both relieve the pain very considerably. If there is retention of urine the catheter must be passed with great care. Mild antiseptics may be injected into the urethra, such as collargol from 1 per cent to 5 per cent, potassium permanganate 1 in 2000 or 1 in 4000, chinosol 1 in 1000. If an abscess forms it should be opened from the perineum and drained by means of a trocar and cannula, with a drainage tube left for irrigation. Internally a laxative should first be given such as a saline or calomel. This may be followed by a course of potassium citrate, combined with tr. of belladonna or hyoscyamus and infusion of buchu. This may be given three times daily. Quinine is also good as an internal antiseptic ; and potassium iodide and potassium bromide are useful in reducing the glandular activity and the consequent pain. The diet must be attended to and should be of a laxative nature. In dogs starchy foods should be avoided. A plentiful supply of water should be provided and drinking encouraged.

Chronic Prostatitis.—This condition is most frequently met with in the dog, and, although it may be a sequel to the acute form, is frequently primary. In old dogs it may be caused by frequent retention of urine and fæces, while on the other hand it may be responsible for those conditions. It is also due in old dogs to excessive sexual excitement, and in other cases to tumour formation.

Symptoms.—In the early stages no symptoms are exhibited until the gland becomes considerably increased in size, except when it is secondary to the acute form. It generally gives rise to frequent urination or to incontinence of urine, while in other cases the enlarged gland presses upon the urethra causing retention of urine and straining for some time before urine can be passed. In dogs the enlarged gland may also cause pressure upon the rectum, bringing about retention of fæces, which may become very aggravated and lead to paralysis or partial paraplegia. Examination per rectum reveals the enlarged gland in the pelvis, but in those cases where the gland is very much enlarged it may fall forward over the brim of the pelvis into the abdominal cavity. The writer has observed enlarged prostates in this situation in the dog approximating the size of one's double fist.

Treatment.—A smart purgative should first be prescribed and a copious enema administered to empty the bowel, which must be kept in a relaxed condition subsequently either by the diet or by the repeated administration of laxatives. If urine is retained a catheter may be passed with care, or in the smaller animals relief may be afforded by the use of the trocar and cannula in the prepubic region. Excision of the prostate in the dog is not to be recommended owing to its difficulty of manipulation. Better results are obtained by castration. When the testicles are removed the prostate undergoes atrophy, and this is accelerated by the internal administration of potassium iodide. If castration is objected to a course of potassium iodide and potassium bromide in combination may afford some relief. In the human subject vaccines are frequently recommended in the treatment of chronic prostatitis due to the gonococcus, but as this organism is never responsible for chronic prostatitis in the dog, and no definite specific cause is known, vaccination is of no service. G. H. W.

DISEASES OF THE CIRCULATORY SYSTEM

These are conveniently considered under three headings :

(1) Diseases of the pericardium.
(2) Diseases of the heart.
(3) Diseases of the blood-vessels.

Diseases of the Pericardium

Pericarditis (Inflammation of the Pericardium).—Pericarditis is not very commonly met with in domesticated animals. It occurs most frequently in cattle, and then it is in the majority of cases due to traumatic causes. It is next most commonly met with in the dog and then in the horse and pig. Pericarditis may be studied under two headings, namely:

(1) Idiopathic pericarditis.
(2) Traumatic pericarditis.

Idiopathic pericarditis is that form which is not associated with a wound. This is the common form in other domesticated animals than the ox.

Traumatic pericarditis is that form in which the inflammation is secondary to mechanical injury, such as the passage of a foreign body, for example a nail, from the reticulum of the ox to the pericardium, or the less frequent puncture of the pericardium through the chest wall.

Etiology.—The cause of pericarditis is probably always bacterial. The organisms may gain access by way of the blood-stream or lymphatics, or from the pleura or through the wound mentioned. Pericarditis is frequently present as a complication of certain specific diseases, such as equine pneumonia, Cape horse-sickness, strangles, and tuberculosis in the horse; and tuberculosis, rinderpest, and contagious pleuro-pneumonia in cattle. In sheep it occurs in the condition known as heart-water in South Africa. In swine it may complicate swine fever, swine plague, and swine erysipelas, while in the dog it may be met with in the course of distemper, and more rarely in tuberculosis.

Idiopathic Pericarditis. — Idiopathic pericarditis may be *acute* or *chronic*.

Acute Pericarditis.—Symptoms.—Frequently the first symptom noticed is some degree of breathlessness, resulting partly from the imperfect contractions of the heart, and in part from the collection of liquid in the pericardial sac, bringing about pressure on the lungs. The chest wall is more or less fixed, but not so much as in pleurisy. There is evidence of pain on pressure on the left side, and frequently muscular twitchings may be noticed corresponding with the heart-beats. The temperature is usually elevated from 2° F. to 4° F. The pulse is hard and frequent, and somewhat irritable in character. Pressure of the liquid on the auricles causes a jugular pulse, and a tendency to œdema and anasarca, particularly under the sternum in front of the chest and the fore limbs. With this disease the larger animals such as the horse and the ox rarely lie down, but if they do it is usually on the right side, and when standing it is a very common thing to have the left fore limb somewhat advanced. On auscultation in the early stages friction sounds may be heard corresponding with each heart-beat and limited to the region of the heart. This is the so-called dry stage, and is followed by the pouring out of an inflammatory exudate into the pericardial sac. At this time the friction sounds become lost and the movement of liquid may be heard in some instances with what has been described as a "metallic" sound. When the fluid is copious the normal heart-sounds become obscured. The pain which was more or less acute during the early dry stages, subsides with the onset of effusion.

Palpitation may occur at any stage and may cause sudden death.

Cases of so-called dry pericarditis may also occur. In these instances the pericardium becomes considerably thickened by the deposition of fibrinous lymph or other pathological tissue as in some cases of tuberculosis. In these cases the heart-sounds become intensified. Owing to pressure on the auricles portal congestion and renal congestion are common sequels to pericarditis, with their attendant symptoms.

Chronic pericarditis may follow the acute form, although it may develop slowly and insidiously without having been preceded by the acute stages. The *chief symptoms* are irregularity of the heart, sometimes associated with palpitation and at other times intermittency, general debility, inability to do prolonged work, and rapidly disturbed respirations. The jugular pulse is also usually present, and in the later stages œdema occurs under the chest, in the body cavities and the limbs.

Traumatic Pericarditis.—The most common form of traumatic pericarditis is that occurring in cattle due to a foreign body such as a nail passing from the reticulum to the pericardium.

Symptoms.—In this condition the train of symptoms is somewhat characteristic. They consist firstly of abdominal irregularities, while the foreign body is in the reticulum and before it reaches the pericardium, when they alter in character and become mainly thoracic. The pericardial symptoms are therefore preceded by frequent and intermittent attacks of tympanites with eructations of gases and colicky pains, with a tendency to fixation of the abdominal wall, and mainly thoracic respiration. As the foreign body passes through the diaphragm into the pericardium, organisms accompany it and pericarditis is set up, partly by the foreign body itself, but to a greater extent by the organisms which accompany it. The abdominal symptoms then subside and thoracic symptoms supervene, and from this point symptoms correspond with those of idiopathic pericarditis. In some cases of traumatic pericarditis, however, sudden death occurs without any symptoms having been observed.

Anatomical Changes.—In the majority of cases of pericarditis a considerable quantity of fluid accumulates in the pericardial cavity and varies in character. It may be clear or it may be turbid and contain a quantity of fibrin which may adhere to the pericardium, causing it to become very thick, sometimes even amounting to an inch in thickness. In older cases new fibrous connective tissue may be produced under the coagulated fibrinous lymph. When due to streptococci the exudate is thick and creamy. In chronic cases there may be simply roughening of the serous membrane and small villus-like projections, while in other cases fibrous adhesions may occur. In traumatic pericarditis of cattle the exudate is almost invariably offensive in odour, and the heart wall itself may actually be pierced by the foreign body. The pericardium is also firmly adherent to the diaphragm, which in turn is also adherent to the reticulum. The fistula or track between the reticulum and the pericardium may also be discovered.

Treatment of Idiopathic Pericarditis.—Absolute rest and quietness are essential, and attention must be paid to the diet, which should be of a somewhat laxative character; in horses and cattle, green food or steamed hay of good quality, boiled linseed and boiled oats. In carnivora, meat extracts and milk and egg should be given. Hot cloths applied to the chest wall will frequently afford relief, while in other cases cold applications may be preferred. Some practitioners prefer to apply a mild blister over the cardiac area. Medicinally internal antiseptics and cardiac tonics are indicated. In the horse and ox the following may be given: potassium iodide ʒii., quinine sulphate ʒi., pulv. digitalis ʒss., administered in ball twice daily. Caffein gr. xx. or camphorated oil may be injected subcutaneously. Tincture of aconite and salicylates are also sometimes used, but as they are very depressing to the heart, particularly the former, they are not to be recommended. In the smaller animals the same agents may be employed in corresponding doses. In those cases where palpitation occurs, a course of potassium bromide may be administered, while if syncope threatens ether or atropine may be administered hypodermically. In cases where there is much dyspnœa due to effusion, the pericardium should be punctured with the trocar and cannula on the left side between the fifth and sixth ribs about the level of the point of the elbow or an inch above it.

In cases of traumatic pericarditis in cattle treatment is not advisable and slaughter is recommended as early as possible.

Treatment of chronic pericarditis also is not worth considering in the case of the horse, since it is doubtful whether he will be restored to a useful condition, while in cattle slaughter is advisable as early as possible with a view to saving the carcase for food purposes. In the case of smaller animals, the dog and cat, treatment is only justifiable from a sentimental aspect of the case.

Dropsy of the Pericardium (Hydrops Pericardii).—Dropsy of the pericardium is not very common, but it may occur as the result of cardiac debility and valvular disease, and also in obstructive lung diseases. It is also present in cases of chronic anæmia, as in cases of wasting parasitic affections, liver fluke, etc. A similar condition occurs in certain specific diseases, as Cape horse-sickness and heart-water. The writer has also met with it in sheep dead of asphyxia, associated with pulmonary changes as the result of dipping immediately after being hurriedly driven. (Out of twenty yearling rams so dipped four died, and from four to six ounces of fluid was found in the pericardium of each of them. The condition to all intents and purposes was drowning.) The liquid is generally clear and limpid, rarely turbid or blood-stained. The *symptoms* of dropsy of the pericardium correspond with those of fluid in pericarditis, and are mainly mechanically produced. There is an absence of all acute symptoms such as pain and fever, and the primary causes of the condition can usually be discovered.

Treatment.—This must depend upon the cause. Anæmia from various causes must be treated, as must any cardiac defects. If dyspnœa becomes excessive puncture of the pericardium must be resorted to. As in the case of dropsy of other serous cavities, however, the condition is most likely to be recurrent, and it is doubtful whether treatment will be justified by results.

Diseases of the Heart

Myocarditis (Inflammation of the Heart Muscle).—Inflammation of the heart muscle is not a very common condition, but when it occurs it is always due to an irritant, the exact nature of which is frequently obscure. *Acute myocarditis* and *chronic myocarditis* are recognized.

Acute Myocarditis.—This condition is usually due to organisms which have been arrested in the capillaries of the heart muscles, and which frequently give rise to multiple abscesses in the heart wall. The organisms may have invaded the blood-stream from any part of the body where suppuration exists, such as strangles, pneumonia, glanders, bacillary necrosis, metritis, and disintegrating thrombus in the umbilical vessels in very young animals. Myocarditis may also occur in the course of any acute septicæmia, and in poisoning by certain agents such as mercury and phosphorus. In acute soya-bean poisoning, small hæmorrhages are produced in the heart muscle (hæmorrhagic myocarditis).

Symptoms.—Symptoms of acute myocarditis are not diagnostic and are chiefly those of irregularity of the heart's action. In some cases palpitations are brought about, while in others intermittence in the heart-beats is a prominent feature. The pulse is increased in number, but is often feeble, thready, and intermittent. The mucous membranes are dusky or cyanotic, and the superficial veins somewhat distended. Sudden death may occur in any animal affected with myocarditis.

Treatment.—Owing to the difficulty of accurate diagnosis treatment is mainly directed to the alleviation of symptoms. A course of digitalis, strophanthus, or squills may be administered, while any suggestion of collapse should be treated with hypodermic injections of ether or camphorated oil. Attention must be paid to the diet and the bowels must be kept in a free condition. Complete rest and freedom from exertion must be enforced.

Chronic Myocarditis.—This form may be a sequel to acute myocarditis, or it may be primary. A not infrequent cause in cattle and occasionally in dogs is tuberculosis, in which case it is usually preceded by pericarditis. The presence of parasites in the heart muscle may also be responsible for the condition, such as trichinella spiralis, cysticercus cellulosæ, cysticercus bovis, echinococcus veterinorum, and sarcocysts.

Symptoms.—These are generally associated with cardiac irregularities, such as palpitation and intermittence, particularly after exercise. Positive differential diagnosis, however, is not possible, and the condition is frequently only recognized on post-mortem examination.

Treatment.—This should be on the same lines as acute myocarditis, and beyond palliating the effects of the condition, treatment is not likely to lead to useful results.

Ossification of the Heart Muscle.—This condition may result from chronic myocarditis, in which a form of cirrhosis of the heart wall has occurred. It is occasionally met with in horses and cattle affecting the right auricle of the heart. There is no satisfactory explanation as to why this portion of the heart should be alone affected. The condition is never diagnosed during life, and although cardiac irregularity is manifest, the true nature of the condition is only established on post-mortem examination.

Endocarditis (Inflammation of the Endocardium).—Endocarditis is usually limited to the endocardium covering the valves of the heart, or to the adjacent areas on the heart wall, or septum.

Two forms of endocarditis may be met with, namely, **acute endocarditis** and **chronic endocarditis.**

Acute Endocarditis.—*Causes.*—This condition is practically always due to bacteria of some kind, and in the majority of cases it is secondary to some bacterial affection elsewhere in the body. In horses it is a fairly frequent disease, and may be a sequel to pyæmia or strangles, or acute rheumatism, pneumonia, and influenza. In cattle it is met with in septicæmic affections, pneumonia, and occasionally tuberculosis. In swine, endocarditis is a very common sequel to swine erysipelas, and it is said to be occasionally present in swine plague. In dogs it may follow distemper, pyæmia, pneumonia, and occasionally tuberculosis.

Symptoms.—The symptoms of acute endocarditis vary considerably and depend to some extent upon the primary disease which it accompanies. They commence with general dulness and depression, fatigue on comparatively slight exertion, with slight elevation of temperature. In other cases debility rapidly develops, associated with spasmodic pain in the chest (angina), muscular twitchings, and indisposition to move. The left fore limb is frequently advanced. The pulse becomes accelerated, short and sharp and irritable, and may be irregularly intermittent. When the aortic semilunar valves are affected the pulse is full and bounding. When the right auriculo-ventricular valves are affected a jugular pulse is noticeable, and this condition is also associated with œdema of the extremities and under the body, and frequently dropsy of the peritoneum as the result of a chronic venous congestion of the liver and other abdominal organs. When the left auriculo-ventricular valves are affected, there is frequently produced spasmodic coughing on slight exertion owing to the blood being dammed back into the lungs. On auscultation of the heart the normal heart-sounds are dulled and not easily identified. They may almost merge into one another and additional abnormal sounds or murmurs may be heard. The heart-beats are also irregular. Palpitation is sometimes set up and there is always the danger of sudden death from syncope. In dogs fainting fits are frequently encountered, particularly in excitable animals. In some patients debility becomes progressive, resulting in weakness of the limbs, particularly the hind limbs, and a rolling gait.

Prognosis.—Prognosis is always grave; in a small proportion of cases complete recovery from acute endocarditis may occur. In a large proportion of cases it is fatal within two or three weeks. In other cases it develops into chronic endocarditis. Another danger which must also be borne in mind consists of the liability of small portions of the valvular growths being carried off in the blood-stream to become arrested as emboli in other parts of the body, causing circulatory disturbances at the seat of arrest, and also possibly setting up a new focus of disease as the result of organisms present in the embolus. In this way endocarditis of the left side of the heart may be followed by lesions in the spleen, kidneys, liver, and muscular and nervous systems, while endocarditis of the right side may lead to secondary lesions in the lungs.

Anatomical Changes.—The lesions are most frequently met with in the auriculo-ventricular valves of the left side and the corresponding valves of the right side of the heart. The semilunar valves are occasionally, but rarely, affected. The common lesions consist of warty-looking growths, so-called vegetations, which are soft in consistence and greyish-white in colour. In horses they may attain the size of the end of a thumb or larger. The growths are generally attached to the contact surface of the cusps, that is, to the surface swept by the blood in passing in the normal direction through the orifice, namely, to the auricular surfaces of the mitral and tricuspid valves, and to the ventricular surfaces of the semilunar valves. This form is generally called a **verrucose endocarditis.** In other cases the so-called ulcerative or malignant endocarditis is met with in which ulcers develop on the valves. In these cases there is great tendency to the breaking up of the vegetations and the formation of emboli. It is possible that in some of these cases the ulcer is produced by the shedding of a small necrotic centre, and acts as a starting-point for the deposition of fibrin and the formation of the so-called vegetations. In more favourable cases ulcers may have healed, leaving only thickened and possibly distorted conditions of the valves, rendering them incompetent.

Treatment.—Absolute rest is essential to success. All forms of excitement and exertion should be carefully avoided. If there is a primary disease present it must be treated accordingly. In addition, cardiac tonics should be administered in conjunction with internal antiseptics. Of the former the best agents consist of digitalis, strophanthus, squills, and nux vomica, while iodide of potassium, quinine, and the salicylates are the best internal antiseptics to use. The salicylates and quinine have the additional advantage of being febrifuge in cases associated with high temperature, while potassium iodide is particularly serviceable in a threefold degree, namely, by its internal antiseptic action, its effect in reducing œdema, and its power of reduction of fibrous growths. In cases where there is danger of collapse stimulants must be prescribed, such as caffein, camphor, ammonia, and alcohol—brandy for preference. A very convenient and easily prepared remedy in such cases consists of hot black coffee, which may be quickly made from coffee extract, together with a little brandy and injected per rectum. This is an instruction which may frequently be left with the owner, who may have need to resort to it in the absence of the practitioner. Ether and camphorated oil are also very useful stimulants, having rapid action when injected subcutaneously. In the case of working-horses a long period of convalescence is required, and the patient should be put through a proper course of conditioning before returning to work.

Chronic Endocarditis (Chronic Valvular Disease of the Heart).—*Causes.*—In the majority of cases chronic endocarditis is the permanent effect of an attack of acute endocarditis, but in some cases it appears to be slow and insidious from the outset. The actual causes, therefore, are the same as in acute attacks of the disease, as in rheumatism, pneumonia, tuberculosis, and swine erysipelas. Chronic endocarditis sometimes develops in connection with chronic endoarteritis, and is sometimes met with in chronic nephritis. It has also been stated that repeated over-exertion is sometimes a cause of chronic endocarditis, but there is very little evidence to support this view.

Anatomical Changes.—The lesions produced in chronic endocarditis especially affect the valves and sometimes the chordæ tendineæ. The valves have become thickened with fibrous tissue, or they carry warty fibrous growths which are never very large. Sometimes the valve is partially adherent to the wall of the orifice, or even to the opposite cusp. In other cases the cusp while still free becomes reduced in size and frequently distorted. In this way either incompetence of the valves (*valvular insufficiency*) or a narrowing of the valvular orifice (*stenosis*) may be produced. As the result of the valvular interference there may be produced also dilatation or hypertrophy of the heart, and chronic venous congestion of the lungs when the left side is affected, and of the abdominal viscera and œdema of the limbs when the right side is affected.

Symptoms of Chronic Valvular Disease.—Before entering into details of the symptoms produced by this condition it is necessary to consider the normal conditions of the heart as observed by auscultation.

During each complete contraction and dilatation of the heart two sounds are produced. The first cardiac sound is a relatively long one, and the second is a short, less voluminous one. They are described phonetically as "lūb-dŭp." They are produced by a sudden closure of the valves during the contraction and dilatation of the heart, the two sides working simultaneously. When the ventricles have completed their contraction, which is called *systole*, they commence to fill again with blood from the auricles through the respective auriculo-ventricular orifice. On completing the dilatation, or *diastole*, the ventricles commence to contract, and this action closes the auriculo-ventricular valves (mitral or bicuspid on the left side and tricuspid on the right side) and produces the first portion of the normal heart-sound (lūb). On completing its contraction and commencing to dilate the semilunar valves which guard the aorta on the left side and the pulmonary artery on the right side are suddenly closed to prevent regurgitation into the ventricles. This sudden closure produces the second portion of the heart-sound (dŭp), which is shorter and sharper and less voluminous than the first portion.

If from any cause any of these valves are incompetent and fail to close, regurgitation is permitted, and the sound which should be produced at the valve in question is either absent or muffled, and is immediately followed by an abnormal murmur (*bruit*) caused by the blood being driven back through the orifice. On the other hand, when the valvular orifice is narrowed or stenosed, an abnormal murmur is produced as the blood is flowing through in the normal direction. This is called a *bellows* murmur and is produced immediately before the portion of the heart-sound occurring at that valve. Thus abnormal murmurs due to incompetence of valve immediately follow the normal sound of that valve, while murmurs due to stenosis precede the normal sound of the valve.

Hence *incompetence or insufficiency* of the auriculo-ventricular valves results in a reduced first sound (lūb) immediately followed by a murmur, while incompetence of the semilunar valves produces an abnormal murmur immediately after the second normal sound (dŭp).

On the other hand, *stenosis* or narrowing of the auriculo-ventricular valves produces a murmur immediately preceding the first normal sound (lūb), and stenosis of the semilunar valves causes a murmur immediately preceding the second normal sound (dŭp). If one auriculo-ventricular valve only is stenosed the effect also is to retard the filling of the ventricle, and the contraction of that side is thus delayed, and the two sides being out of unison a duplicated first cardiac sound is produced. If one semilunar valve only is stenosed, there may similarly be a duplication of the second cardiac sound.

Abnormal murmurs produced during systole are called *systolic murmurs*; similar sounds during diastole are called *diastolic murmurs*. Systole corresponds with that portion of the normal heart-sound from the commencement of the first cardiac sound (lūb) up to the commencement of the second cardiac sound (dŭp). Systolic murmurs, therefore, are any abnormal sounds produced during that period, and may be produced by incompetence of the auricular ventricular valve or stenosis of the semilunar valve.

Diastole is the period from the commencement of the second heart-sound (dŭp) to the commencement of the succeeding first heart-sound (lūb), and diastolic murmurs thus occur during that period. They may be produced, therefore, by incompetence of the semilunar valves or stenosis of the auriculo-ventricular valves.

Now to consider each valve and valvular orifice separately.

1. **Stenosis of the Left Auriculo-ventricular Orifice; Mitral Stenosis.**—In *mitral stenosis* there is a narrowing of the left auriculo-ventricular orifice which causes an impediment to the flow of blood from the left auricle into the ventricle during diastole. As the result of this a diastolic murmur is produced, occurring generally towards the end of diastole, that is, immediately before the production of the first sound (lūb) produced by systole. This is sometimes spoken of as a *presystolic murmur*. The murmur varies in intensity and usually increases in volume until it runs almost into the first cardiac sound. The effect of the stenosis may be to produce dilatation of the left ventricle, and this is often followed by hypertrophy both of the ventricle and auricle. This condition may result in a duplication of the second cardiac sound. The reduced rate of flow of blood from the auricle brings about some degree of hyperæmia of the lungs and consequent breathlessness on exertion, and ultimately leads to dilatation and hypertrophy of the right ventricle. The pulse in these cases is smaller and softer than normal, since the left ventricle drives a smaller volume of blood into the arteries.

2. **Stenosis of the Right Auriculo-ventricular Orifice; Tricuspid Stenosis.**—Stenosis of this valve results in the production of a presystolic murmur in the same manner as in the case of mitral stenosis, and in a similar manner it may produce some slight hypertrophy of the right auricle and ventricle. The pulse is small and feeble and there is a tendency to stasis of the vena cava and the veins leading to it. This may result in general dulness and depression, breathlessness on exertion, a slight jugular pulse, and sometimes to general œdema. This latter fact may assist in differentiation of tricuspid stenosis from mitral stenosis.

3. **Incompetence of the Bicuspid or Mitral Valve; Mitral Insufficiency.**—Incompetence of the left auriculo-ventricular valves may be brought about as the result of distortion of one or both cusps or to excessive dilatation preventing the complete closure of the orifice during systole. The effect of this is to produce a

muffled first cardiac sound and a systolic murmur which immediately follows the first cardiac sound. The intensity of this murmur is usually softer than the presystolic murmur of stenosis. The second cardiac sound is usually intensified. The effect of this condition on the lungs is to produce dyspnœa with spasmodic coughing and accelerated respirations as the result of regurgitation into the pulmonary veins and lungs. In the horse this may sometimes be confused with broken-wind, while in the dog the cough often closely resembles that of asthma and is often spoken of as cardiac asthma. This pulmonary stasis raises the pressure of blood on the right side of the heart, and frequently leads to dilatation or hypertrophy of the right ventricle and often a resulting jugular pulse and general œdema. The arterial pulse is fairly full and regular, but on excitement it may become irregular and intermittent.

4. **Incompetence of the Tricuspid Valves; Tricuspid Insufficiency.**—This condition may be brought about by similar pathological conditions as mitral insufficiency, and is similarly responsible for a soft systolic murmur which can be best heard when the left fore limb is drawn well forward. Each heart-beat is also accompanied by a well-marked jugular pulse, and breathlessness or dyspnœa on exertion. The condition is also responsible for a general chronic venous congestion resulting in œdema of the tissues and body cavities, particularly ascites. In many cases fainting fits also occur, and in the horse this is responsible for many attacks of vertigo and so-called "sleepy-staggers" as the result of venous congestion of the brain. This effect may occur in any animal when at exercise. The patient may suddenly become erratic in its movements and plunge and fall, and become partially unconscious. After remaining on the ground a short time consciousness returns and the patient rises somewhat dazed, but soon apparently completely recovers. The well-marked jugular pulse together with the pronounced œdema assists in distinguishing tricuspid insufficiency from mitral incompetence.

5. **Stenosis of the Aortic Orifice.**—In narrowing of the aortic orifice an intense and prolonged systolic murmur is produced, that is, immediately after the first cardiac sound and running almost into the second cardiac sound. The second cardiac sound is reduced in intensity and may be duplicated owing to prolonged systole. The pulse is smaller and slower than normal, taking a longer time to pass under the finger, but is usually firm and regular. The extra work thrown upon the left ventricle leads first to dilatation, which is generally followed by hypertrophy, and in some instances the impulse of the heart against the chest wall may be heard without direct auscultation.

6. **Stenosis of the Pulmonary Valves.**—In this condition, *which is of very rare occurrence*, a systolic murmur is produced as in stenosis of the aortic orifice. It results in dyspnœa, breathlessness on exertion, and sometimes cyanosis. It is distinguished from aortic stenosis mainly by the character of the pulse being unaltered.

7. **Incompetence of the Aortic Valves; Aortic Insufficiency.**—In incompetence of the aortic valves regurgitation is permitted into the left ventricle during diastole, thus producing a diastolic murmur which is comparatively soft and immediately follows a reduced second heart-sound. This murmur is distinguished from the diastolic murmur or presystolic murmur of mitral stenosis by its reduced intensity, particularly towards the completion of diastole. The condition results in hypertrophy of the left ventricle as the result of which the pulse is strong and bounding, and is sometimes described as a "water-hammer" pulse in human medicine. In the sphygmographic tracing the dicrotic notch is wanting owing to the imperfect closure of the aortic semilunar valves. The impulse of the hypertrophied heart on the chest wall is usually very pronounced. In bad cases the respiration may be interfered with, resulting in dyspnœa on exertion, in some cases in horses somewhat resembling broken-wind.

8. **Incompetence of the Pulmonary Valves; Pulmonary Insufficiency.**—This condition is *very rarely* met with. In insufficiency of the right semilunar valves regurgitation is permitted into the right ventricle during diastole, resulting in a diastolic murmur as in the case of aortic insufficiency. The murmur is less pronounced than in the latter case. The pulse is not altered in character, and this fact serves to differentiate the condition from incompetence of the semilunar valves of the left side of the heart. The effect on the respiration is to cause dyspnœa and general breathlessness on slight exertion.

Prognosis of Chronic Endocarditis.—In chronic valvular disease of the heart prognosis should always be guarded. In a general way, however, it is unfavourable in so far as it refers to recovery. Disease of the *aortic orifice and valves* is likely to be the least serious, owing to the strength of the left ventricle which overcomes the obstruction for a considerable time. Disease of the *mitral valves* is more serious, owing to the great interference with the circulation through the lungs and its effect on respiration and, through the lungs, on the right side of the heart. Diseases of the *right side of the heart* are very unfavourable, owing to the inability to develop compensatory hypertrophy, and also the very great tendency to the production of chronic venous congestion both of the liver and other abdominal organs, and of the brain, leading to vertigo and other cerebral symptoms.

In the case of the horse the question of his ability to undertake further work is a matter of first importance. Although there is always the possibility that sudden death may occur as the result of any of these forms of chronic endocarditis, yet it is an undeniable fact that considerable numbers of horses, given reasonable care and attention and not overworked, may remain useful for slow work for a considerable time. It is the case that a considerable proportion of horses diagnosed as being bad roarers are in reality affected with chronic heart disease.

Dogs with chronic cardiac lesions may live for years without any serious inconvenience, but there is always the possibility that any undue exertion or over-excitement may result in sudden death.

Treatment.—The treatment of chronic endocarditis is mainly palliative, since it is impossible to restore the valves and orifices to their normal condition. Over-exertion and excitement must always be avoided and the general functions must be carefully attended to. The diet must be easily digestible and of a demulcent nature in order to obviate any possibility of constipation and undue straining. This may be also attained by the administration of a mild laxative periodically. Stronger purgatives should always be avoided on account of their production of general cardiac depression. Cardiac tonics may be administered such as digitalis, strophanthus, squills, and nux vomica. These agents, used with discretion, tend to improve the force and vigour and the regularity of the heart's action. In cases where syncope threatens, hypodermic injections of caffein or of atropine may be administered. The sequels of the cardiac insufficiency must also be treated as they arise. In the case of dyspnœa and spasmodic coughing glyco-heroin and sulphuric ether may be added to the cardiac tonics. In cases associated with œdema of the cavities and tissues diuretics may be added and the drinking-water curtailed. For this purpose iodide of potassium is particularly useful combined with nitrate of potassium and the cardiac tonics mentioned. Dropsy of the cavities must also be relieved by tapping, and this must be repeated from time to time when necessary.

Hypertrophy of the Heart; Cardiac Hypertrophy.—Cardiac hypertrophy implies an increase in the muscular tissue of the heart. The degree of hypertrophy is estimated by its weight and the thickness of its walls, not by external measurement. The average weight of the horse's heart is from 7 to 8 lbs., but varies according to the size of the horse. It averages probably a little less than 1 per cent of the total weight of the living body. In cattle it is proportionately smaller, being between 0·4 and 0·5 per cent of the living weight; in the dog about 1 per cent of the body weight. A hypertrophied heart may attain twice the normal size of the heart or even more, and hypertrophied hearts in the horse up to 30 lbs. in weight have been recorded.

Hypertrophy and dilatation of the heart are frequently coexistent. It has been called *simple hypertrophy* when unassociated with dilatation, *excentric hypertrophy* when it is associated with dilatation, and *concentric hypertrophy* when it is associated with a reduction in capacity of the heart cavities. The latter condition is by many observers regarded as a post-mortem condition of a firmly contracted hypertrophied heart.

Causes of Cardiac Hypertrophy.—The chief cause of hypertrophy of the heart is increased resistance which has to be overcome, provided the nutrition of the heart wall is maintained. In cases of increased exertion the heart may respond to the extra strain upon it, and according to the degree of resistance hypertrophy and dilatation may be met in varying degrees. It may thus be a physiological condition in excessive muscular exercise or training as in race-horses and hunting-dogs. Pathological hypertrophy is, however, brought about by increased resistance due to a variety of morbid conditions. Hypertrophy of the left ventricle may result from incompetence or stenosis of the aortic valve or from chronic interstitial nephritis which raises blood pressure and increased cardiac resistance on the left side. It may also be due to stenosis or incompetence of the mitral valve, and this usually entails a simultaneous hypertrophy of the left auricle. Hypertrophy of the right ventricle may be brought about by stenosis or incompetence of the pulmonary valves or increased blood pressure in the lungs as results from chronic emphysema. Stenosis or incompetence of the mitral valve may also cause hypertrophy of the right ventricle by producing some degree of pulmonary congestion, and so increasing the work of the right ventricle to overcome it. It is also said that adhesions between the heart and pericardium may bring about hypertrophy of the heart.

Anatomical Changes.—One or both sides of the heart may be affected, the left side is more frequently hypertrophied than the right side, and the ventricles are more frequently hypertrophied than the auricles. When both sides are hypertrophied the heart appears somewhat rounder than normal. When the left ventricle alone is affected the heart appears longer and more pointed than normal. When the right ventricle is affected the heart appears broader than normal, particularly at its base. When the hypertrophied cardiac muscle is cut into it is found to be somewhat firmer than normal heart muscle but not altered in colour.

Symptoms.—In hypertrophy there may be only few symptoms observed, particularly if both sides are involved. When one side alone is affected there is usually a duplication and intensification of the second cardiac sound. When, however, compensatory hypertrophy of the other side occurs the second cardiac sound remains intensified but is not duplicated. When the left heart is affected, and this is the common condition, the pulse is strong and slow, and there is an increased impulse against the chest wall, and the whole body may be seen or felt to shake at each heart-beat.

Treatment.—There is no specific treatment which is directly applicable to hypertrophy of the heart. If cardiac irregularities occur the same lines of treatment must be adopted as for chronic endocarditis, since this condition is in such cases responsible for the cardiac hypertrophy. In the case of the food-animals that are otherwise in a fit condition, slaughter is the best procedure.

Dilatation of the Heart.—As already stated under "Cardiac Hypertrophy," dilatation of the heart is in many instances accompanied by hypertrophy. It is a condition in which there is an increase in size of the heart cavities, and it frequently precedes hypertrophy.

Causes.—Dilatation of the heart is brought about by increased blood pressure without a corresponding response on the part of the cardiac muscle. It may therefore occur under similar conditions as detailed under hypertrophy, but in cases where good muscular tone exists it is rapidly followed by hypertrophy. Dilatation may be brought about by forced exercise when an animal is not in condition, as in the case of hunters overridden in the early part of a season, and it is commonly met with in horses affected with chronic broken-wind (pulmonary emphysema). Dilatation may also occur as the result of fatty degeneration of the heart. In rare instances it is said to be due to the presence of tumours or of parasites in the heart.

Anatomical Changes.—The cardiac muscle in dilatation without hypertrophy is soft and flabby, and when cut the walls are thin and often somewhat pale, while the cavities of the heart are larger than normal. In the case of the auricles extreme thinness may be easily recognizable even without cutting. The chordæ tendineæ are often thin and elongated.

Symptoms.—In simple dilatation of the heart there is always general muscular weakness and inability to do prolonged work. The general strength of the circulation is reduced, and there may be regurgitation through the auriculo-ventricular orifices, resulting in troublesome coughing and dyspnœa on exertion or excitement. Venous engorgement occurs with œdema in various parts of the body, particularly the liver and the abdominal area and limbs. On auscultation systolic murmurs may be observed owing to insufficiency of the auriculo-ventricular valves. There may also be a duplication of the second cardiac sound. Palpitation sometimes occurs and abnormal rhythm may be set up. The pulse is slow and weak. In horses dyspnœa set up is sometimes very pronounced, resulting in audible respiration which is not infrequently diagnosed as roaring, and is sometimes wrongly attributed to left recurrent paralysis of the larynx (*laryngismus paralyticus*). This fact is undoubtedly responsible for a number of the so-called failures of the ventricle-stripping operation for roaring in horses, particularly in those cases which appear to have been improved when first put to exercise two or three months after operation but which rapidly become worse when harder work is resumed. The writer has made careful post-mortem examinations in two such cases in hunters where severe roaring and dyspnœa have recurred after successful operation with apparently excellent results. In each case the only lesion discoverable was dilatation of the ventricles. In his opinion the temporary relief of symptoms after operation was due to the temporarily improved condition of the heart brought about by the enforced rest. When the horses were put to work again the old trouble was renewed.

Treatment.—The treatment of dilatation of the heart is generally only palliative, since it is usually impossible to restore the cardiac muscle to its normal condition. The work of the heart must be diminished, and for this purpose absolute rest and warmth are essential, while the patient receives a generous, easily digested diet. A mild laxative should be administered which at the same time reduces blood pressure and reduces straining in defæcation. Medicinally the same agents should be used as are recommended for chronic endocarditis, which is so often the cause of the condition. Digitalis and nux vomica are particularly valuable. In the smaller animals stimulants should always be at hand in cases of heart failure, such as ether or amyl nitrite or atropine, which may be injected subcutaneously. The cautious inhalation of strong ammonia or smelling-salts may also be resorted to, while rectal injection of hot coffee with brandy is a very useful restorative. All forms of excitement or exertion must be avoided. Any complications set up must be treated as they arise. In the case of horses they may often be put to light, slow work after a few weeks' rest. In the case of the food-animals that are otherwise in a fit condition, slaughter is the best procedure.

Fatty Degeneration of the Heart.—This condition is met with most frequently in the horse and is generally associated with fatty degeneration in other organs, particularly the liver. Deposition of fat under the pericardium may also occur in very fat animals, especially old dogs, and is often associated with an increased amount of fat in the muscle tissue (fatty infiltration).

Causes.—The most frequent causes of fatty degeneration are various bacterial toxins, and it is therefore frequently met with in animals after subacute and prolonged febrile diseases such as strangles, influenza, pleurisy, and empyema. It may also be brought about by chronic poisoning with arsenic, antimony, and phosphorus. Fatty degeneration may also result from malnutrition.

Anatomical Changes.—Fatty degeneration may either be *general* or *local*. In general fatty degeneration the muscle is paler than normal and is greyish-red or yellowish-red in colour and has a somewhat macerated appearance. The heart walls are flabby and easily torn. In the majority of cases dilatation is also present. In local fatty degeneration of the heart the affection is usually limited to a local patch and is probably due to disease of a branch of the coronary artery interfering with nutrition. A local patch of fatty degeneration may be followed by aneurism of the heart wall. In fatty infiltration there is an increase in fat under the epicardium commencing in the furrows of the heart and spreading over the surface of the heart. This condition often accompanies general obesity. Fatty filtration may debilitate the heart, but is not so serious as fatty degeneration.

Symptoms.—There is a general weakness of circulation resulting in a weak, soft, long-drawn-out pulse and general œdema. The animal is easily exhausted and may be the subject of dyspnœa and troublesome coughing and often fainting fits (sleepy staggers or vertigo in the horse). As fatty degeneration usually leads to dilatation of the heart, the same phenomena may be observed on auscultation as described under that condition. Fatty degeneration may lead to sudden death from failure of the heart's action. In other cases it may prove fatal through rupture of the heart. The latter condition is most likely to occur as the result of local fatty degeneration with aneurism.

Treatment.—Curative treatment is not likely to be successful. In the case of horses the same course should be followed as recommended for dilatation of the heart. In the case of the food-animals that are otherwise in a fit condition, slaughter is the best procedure.

Rupture of the Heart.—This condition is most likely to occur as the result of local fatty degeneration. It may also result from great exertion in cases of dilatation and valvular disease of the heart. In some cases rupture is the result of direct injury either through external wounds or in cattle by a foreign body, such as a nail, entering the heart from the reticulum or second compartment of the stomach.

Symptoms.—In rupture of the heart the symptoms are those of rapid internal hæmorrhage and are often preceded by some sudden exertion or a fall. The patient is noticed to tremble violently and intense dyspnœa comes on. The mucous membranes are very pallid. The animal staggers and falls in respiratory convulsions and death follows immediately.

There is obviously no treatment available for rupture of the heart.

Functional or Nervous Diseases of the Heart ; Cardiac Neuroses

Palpitation (Tachycardia).—Palpitation is a nervous condition of the heart in which there is produced an increase in the number and force of the heart-beats, which in some instances may also be irregular. The condition is probably due to increased irritability of the sympathetic nerve and may be brought on by fright, over-exertion, over-excitement, or a fall, and is more likely to be met with in animals affected with valvular disease than in others. It may also follow as a sequel to acute febrile diseases such as influenza and distemper, and debilitating affections such as anæmia, and it may also result from chronic lead-poisoning. Palpitation in other cases may be due to reflex action through the pneumogastric nerve, and in such cases may follow dyspepsia and dietetic errors. In some highly nervous patients the fact of examination by a stranger in many cases may set up attacks of palpitation. This is particularly the case in excitable breeds of dogs, sometimes in horses, and less so in cattle.

Symptoms.—The symptoms usually develop suddenly. The patient is seen to be in a state of anxiety, often trembling and wearing a distressed facial expression. The impulse of the heart against the chest wall may be observed in mild cases, while in severe cases the whole body may be seen to throb at each heart-beat, while the heart-beat is tumultuous and may be heard some distance away. The arterial pulse is frequent and hard but as a rule small, sometimes intermittent. A jugular pulse is often present. After a while the symptoms may subside, only to be reproduced if the primary cause is repeated or not removed. The duration of the attack may be from half an hour to half a day in the horse and ox, whereas in the dog the duration is generally much shorter. In the latter animal one should bear in mind that in nervous individuals acceleration of the heart is a common occurrence when they are being examined by a stranger. Such a condition, however, should not be regarded as palpitation or tachycardia.

Treatment.—Rest and quietness, with freedom from excitement or exertion, are essential. Treatment must depend to some extent on the discovery or otherwise of a primary cause. The paroxysms may be checked by the administration of morphine or potassium bromide or chloral hydrate, and the spasms may be cut short by cautious inhalation of amyl nitrite. When the condition is associated with cardiac debility, as after influenza or distemper, sedatives should be followed by a course of cardiac tonics such as digitalis, strophanthus, strychnia, or arsenic. In those cases where it is secondary to dyspepsia or dietetic errors a mild purgative should be administered and the diet must be attended to and consist of concentrated, easily digested foods, as far as possible free from excessive indigestible fibre. In the case of herbivora boiled linseed and good oats with good quality hay are essential, while in carnivora a meat diet should be prescribed.

Bradycardia (Slow Heart's Action).—This is a condition of abnormally slow action of the heart, and is due to a functional disturbance without any recognizable structural change. It may occur during convalescence from acute affections such as influenza and distemper, whereas in other cases it may be secondary to gastric and other alimentary disturbances. A slow action of the heart also occurs in certain structural affections such as fatty degeneration of the heart, dilatation of the heart, myocarditis.

Symptoms.—Animals with this affection are generally sluggish in their movements and are frequently the subjects of other cardiac irregularities, while in some cases affected animals may appear quite well although they may be unable to undergo prolonged severe exertion. The heart sounds are usually clear and distinct but less frequent than normal. In the horse their number may be reduced to twenty or less per minute, while in the dog it may be reduced to twenty or twenty-five per minute. The history of the case also assists one in arriving at a diagnosis, particularly when due to a previous attack of a debilitating disease.

A condition of " slow heart-beat " is not infrequently met with in certain types of horses with puffy limbs and weak superficial circulation, the so-called " gummy-legged " horses. In these animals the heart's action is reduced distinctly below normal. These are examples of bradycardia, but are not exaggerated cases.

Treatment.—In pronounced cases the action of the heart can be improved almost immediately by the hypodermic injection of small doses of atropine or ether. Following this, a course of cardiac tonics gives the best results, such as small repeated doses of strychnia and digitalis together with ammonia. If the condition is secondary to alimentary disturbance, a mild laxative should also be given, and the diet should be concentrated, easily digestible, and not too bulky.

Cardiac Arrhythmia.—Two forms of cardiac irregularities or interference with rhythm may be met with:

(1) Intermittent systole (cardiac intermittence).

(2) Extra systole.

The former is the more common condition in the lower animals; the converse is generally held to be the case in the human subject.

Cardiac Intermittence. — On examining the heart or pulse of an animal it may be noticed that a beat is occasionally missed. This abnormal interval between the heart-beats is called an intermittence. Intermittence may be regular or irregular in its occurrence, that is to say, it may occur at regular intervals (regularly intermittent) or at irregular intervals (irregularly intermittent). In the latter case also the duration of the intermittence may vary, thus producing a much more serious condition, since the pause may become too prolonged and result in syncope. The regularly intermittent pulse in veterinary patients is rarely very serious.

Causes.—Cardiac intermittence or omission of ventricular contractions is a fairly common condition in the horse and is frequently a sequel to acute affections such as influenza. In other cases it is secondary to indigestion, and is then brought about by reflex action through the vagus nerve. In the dog cardiac intermittence is an extremely common condition, particularly in highly nervous animals when they are being examined. In such cases it is of small significance as an indication of disease. In any species intermissions may occur as the result of heart diseases such as pericarditis, myocarditis, and endocarditis.

Extra Systole.—This is a condition in which a small pulse-wave may be appreciated by the finger just before the normal pulse is due. It is a kind of double pulsation, and, as in the case of intermittence, it may be regular or irregular in its occurrence. This extra pulsation can be more easily felt at the radial artery than at arteries nearer the extremities. The irregularity may also be observed on auscultation. The condition is generally due to some structural cardiac disease such as valvular disease, but it may also be of nervous origin.

There may also be observed a form of cardiac irregularity which does not fall into either of the preceding classes. In this condition the heart beats more or less irregularly with a varying intensity or impulse, and with irregular intervals which rarely or never extend over the period of one heart-beat. The condition is common in old horses, and is usually due to general debility, often brought about by overwork and under-feeding. It may also occur during convalescence from debilitating affections.

Treatment of Arrhythmia.—In the case of a regularly intermittent heart little treatment is called for. An affected horse may perform his usual work with little or no inconvenience. On the other hand, in cases of irregular intermission or extra systole rest is essential. The primary cause where possible should be ascertained and treated accordingly. When the irregularity is due to a primary heart disease some temporary improvement may be obtained by a course of digitalis and strychnia. In those cases that are secondary to alimentary disturbance, as dyspepsia or constipation, a mild laxative should first be administered, but not a violent purgative, and a course of combined alimentary and cardiac tonics administered such as digitalis, nux vomica, ammonia, and carbonate of iron. In addition, in the larger animals a small dose of linseed oil ℥ii.-iv. may be given daily in the food with advantage, while in the smaller animals cod-liver oil and malt combined with compound syrup of phosphates is often serviceable.

Angina Pectoris.—This is a term applied to distressing and painful paroxysms arising from some affection of the heart. Its etiology is somewhat obscure, and the condition is rarely diagnosed in the lower animals, owing to the absence of the subjective symptoms. It is probably due in the majority of cases to some structural heart disease such as valvular affection, and more rarely to disease of the coronary arteries. A similar condition sometimes occurs in dogs, particularly in the Far East, as the result of a thread-worm, the *Filaria immitis*, which may be met with in the right ventricles.

Symptoms.—These may be brought on by excitement, exertion, or a sudden unexpected movement. In the case of the horse, he may suddenly pull up and cannot be persuaded to proceed for some minutes. Temporary relief occurs, but recurrence takes place at very short intervals. When standing, the left fore limb is usually advanced and lameness is suspected. On auscultation the heart is often palpitating, but the pulse may be small and irregular. Convulsions and twitchings of the muscles and the chest wall, and the neck, may also be observed. Attacks may be brought on in susceptible subjects at irregular intervals by exercise, and may terminate fatally from syncope.

Treatment.—Immediate relief may be afforded by inhalation or hypodermic injection of amyl nitrite, and this should be followed by cardiac sedatives, such as morphine or chloral hydrate, or a general anodyne to relieve the pain, such as bromide of potassium or ammonium, which has little or no cardiac depressant action. Excitement and exertion must be carefully avoided.

Diseases of the Blood-Vessels

Arteritis; Artereitis

Inflammation of the arteries is a comparatively rare disease in the lower animals except when due to traumatic causes or associated with parasites. The most common instance of the condition occurs in the horse as the result of infection with the *Strongylus vulgaris*, which also sets up aneurism and thrombosis. In *atheromatous arteritis* a degeneration occurs involving the intima as the result of which a small patch may become detached, leaving a shallow erosion or ulcer which may act as the starting-point of a thrombus. At other times it undergoes calcification. The commonest seats of atheroma are the aorta and the large arteries leading from it.

Symptoms.—There are no symptoms exhibited during life by which arteritis in the lower animals can be detected, and it is only discovered on post-mortem examination.

Aneurisms

An aneurism is a local dilatation of an artery, generally brought about by a weakened condition of the arterial wall following either arteritis or degeneration. The commonest form of aneurism is brought about by the *Strongylus vulgaris* in the horse. The larvæ of this parasite are carried to the vasa vasorum, and there increasing in size produce the lesion of the intima and general weakening of the vessel wall which is followed by dilatation and thrombus formation. The common situation is the large mesenteric artery or one of its branches. This condition sets up a violent verminous colic in the horse. (See page 304.)

Thrombosis of the Iliac Arteries in the Horse.

(See Surgical Section, p. 735).

Phlebitis (Inflammation of the Veins)

The commonest cause of this condition is the presence of bacteria, which may gain access through a wound or may penetrate through diseased tissue in its immediate neighbourhood. The veins may thus become involved in the lungs from pneumonia, and in the liver from the portal vein, and in the womb from metritis. Phlebitis may also follow as a sequel to the operation of phlebotomy in "bleeding." Almost invariably at the seat of phlebitis a thrombus is produced. This causes a venous congestion with œdema of the area drained by the affected vein.

Varices ; Varicose Veins ; Phlebectases

A varicose vein or varix is the dilatation of a vein, and may be uniform or irregular. It is generally brought about as the result of some obstruction to the venous circulation in areas where there is very little support to the veins. The condition is extremely rare in the lower animals. It is most frequently seen in the spermatic veins. When it occurs in the lower portion of the rectum it is called "hæmorrhoids," a condition which is extremely rare in the domesticated animals.

Parasites of the Heart and Blood-Vessels

Filaria immitis (Filaria hæmatica. Dirofilaria immitis).—This parasite is met with frequently in dogs in China and Japan and Panama. In Europe it is seen in Italy, but otherwise only in animals imported from those countries or amongst mariners' pets that have visited those waters on board ship. The parasites are more correctly regarded as hæmatozoa or blood parasites, but the sexually mature worms are met with in the cavities of the heart, more particularly on the right side and only occasionally on the left side. They may be present in varying numbers and may be very numerous, rolled up in tangled masses and often involving the chordæ tendineæ and the valves. The embryos, which are microscopic in size, invade the general circulation.

The adult parasites are long white worms, the male being about 6 or 7 inches long and possessing a spiral tail. The female is from 10 to 15 inches long and is ovoviviparous, *i.e.* gives birth to living embryos. The embryos are carried off into the blood-stream, where they may be found in the peripheral vessels, particularly at night time. There are remarkably few, and they are difficult to find in this situation in the day time. They are transmitted from dog to dog only by means of mosquitoes or gnats (culicidæ). Affected bitches have been stated to infect their puppies before birth through the placenta.

Symptoms.—Many affected dogs show no symptoms, but in the majority of cases the parasites set up a fatal illness. The principal symptoms are those of chronic endocarditis—dulness, breathlessness, general loss of condition and flesh, and progressive emaciation. A distressing cough may be set up, partly by the interference with the heart's action and partly through embolism of the pulmonary blood-vessels. Dropsy of the cavities and tissues may occur, and in some cases hæmaturia or blood-stained urine is set up. In some instances muscular pains are produced of a flitting or metastatic character, suggestive of muscular rheumatism, and sometimes great weakness of the hind limbs amounting almost to paralysis. Brain symptoms may also be set up resembling epilepsy, while in other cases angina pectoris may be met with. Occasionally skin lesions are produced with small irritating papules in which embryos of the parasite may be found. The heart becomes hypertrophied, and convulsions may be set up either of asphyxia or syncope. Positive diagnosis can only be established by microscopic examination of blood taken at night.

Treatment.—Medicinal treatment is not very hopeful. Atoxyl and other arsenical preparations have been recommended, and though they may reduce the number of embryos present it is doubtful if they have any effect on the adult parasites in the heart. Cardiac and respiratory convulsions, and also brain symptoms, may be relieved by bromides. The general condition may be maintained by cardiac tonics and a generous diet, including cod-liver oil, malt, and iron emulsion. If there is any skin irritation creosote and calamine lotion may be applied.

Preventive measures consist in protecting the dogs from mosquitoes by keeping them indoors after sunset, and a campaign against the insects and their breeding-places where accessible. Fumigation of kennels may also be of service.

Strongylus vasorum.—This is a round worm met with in dogs, principally in the south of France and Italy, pinkish white in colour, the male up to ½ inch in length and the female up to ¾ or ⅘ of an inch. It is met with in the right side of the heart and the pulmonary artery, often very numerous and collected into small bundles about the size of peas. The eggs are carried off into the pulmonary circulation and embryos are developed in the lungs. It is believed that they are then expelled by coughing and may be taken up by other dogs.

Symptoms.—The chief effects are those of endocarditis and thrombosis of the pulmonary vessels. There may be a cough, progressive emaciation and anæmia, irregular pulse and heart, and inappetence. The right side of the heart may become hypertrophied and some dropsy may result. The condition is almost impossible to diagnose and it is rarely fatal. Its true nature, however, may be established by the discovery of embryos in the nasal discharge, or in outbreaks by destroying an affected dog and making a careful post-mortem examination.

Treatment.—Not very hopeful, but good results have been claimed from the use of turpentine in doses of ʒss.-ʒi. given daily either in emulsion or made into a pill.

Parasites of the Heart Muscle

The **cystic stage** of several **tapeworms** may be met with in the heart muscle, but they do not give rise to any symptoms by which their presence can be diagnosed during life. They are elliptical in shape and their average length is from ⅙ to ¼ of an inch—that is, about the size of barley grains. They are in reality parasites of the general muscular system and will be dealt with under that section. Beyond naming them no further reference is necessary at this point. They are as follows :

In the ox : *Cysticercus bovis*, bladder-worm of the unarmed tænia (*T. saginata* or *medio-canellata*) of man.

In the pig : *Cysticercus cellulosæ*, bladder-worm of the armed tænia (*T. solium*) of man.

In the sheep : *Cysticercus ovis*, bladder-worm of the *Tænia ovis* of the dog.

Sarcosporidia are not infrequently met with in the heart muscle in common with the muscular system in general. They may infest most species of animals, and are the commonest in the sheep (*Sarcocystis tenella*) and in the pig (*Sarcocystis miescheri*). They have also been observed in the heart of the ox and goat and more rarely in the horse and dog. In the pig the sarcocysts can just be seen by the naked eye, being from $\frac{1}{12}$ to ⅛ of an inch in length and producing a granular appearance of the heart muscle. In the sheep they are smaller than in the pig and are rarely visible to the naked eye.

Sarcosporidia are not known to cause any symptoms during life by which their presence in the heart muscle can be diagnosed or suspected

Parasitic Aneurisms

Nematode Worms.—The walls of the larger blood-vessels are the normal habitat of certain nematode worms belonging to the genera Eleophora, Onchocerca, Spirocerca, and Strongylus. In the cases of the first three genera the worms invade the aorta and attain maturity in that situation, while in the strongylus it is an immature stage of the worm that inhabits the vessel wall, usually the great mesenteric artery. Their presence so weakens the vessel walls that aneurism very frequently results. The symptoms set up vary with the seat of the aneurism, but are most serious, owing to the greater frequency and wide distribution of the parasite, in the case of strongylus (see "Verminous Colic," p. 304).

The following species are known to be responsible for aneurisms :

Eleophora poeli in cattle : Malay States.

Onchocerca armillata in cattle : India, Sumatra, and N. Africa.

Spirocerca (*Spiroptera*) *sanguinolenta* in dogs : Mediterranean, Europe, Far East, Brazil.

Strongylus vulgaris in horses : world wide.

G. H. W.

CLINICAL PATHOLOGY OF BLOOD

The blood may be considered as a tissue, composed of cellular and intercellular constituents, the former being the red and white corpuscles and the platelets, and the latter the plasma.

Owing to its coming into intimate association with all other tissues, its examination may afford useful information as to the state of the animal as a whole ; also affording particular information in the case of diseases when the causal organism or the more definite lesions are present in the blood.

Methods of Examination

The ordinary blood-examination consists of counting the red and white corpuscles, estimating the amount of hæmoglobin, and examining stained blood-smears. In special cases the plasma is tested for specific antibodies, *e.g.* agglutinins.

Procuring the Blood.—Small quantities may be drawn from any superficial vein where the circulation is normal, the ear veins being most convenient ; in birds, the comb or under the wing.

The chosen site has the hair clipped, and is washed and dried. The vein is cleanly incised, and when the blood flows the first few drops are wiped away.

Before making the incision any required apparatus should be ready to hand—slides, pipettes, and diluting fluid, etc.—so that the required manipulations may be carried out before the blood has time to clot.

If the plasma is to be examined for agglutinins it will be necessary to collect a few c.cm. of blood in a sterile bottle. This is most easily done, in the larger animals at any rate, by drawing blood through a hypodermic needle from the jugular vein.

Counting the Red Corpuscles

The red corpuscles are so numerous that they must be diluted before counting, and for this process a fluid must be used which will not allow the blood to clot and will not injure the corpuscles. Hayem's Fluid combines these qualities and consists of :

Mercuric chloride 0 5 gm.
Sodium chloride 1 gm.
Sodium sulphate 5 gm.
Distilled water 200 c.cm.

The most generally used instrument for combining the processes of dilution and counting of the red corpuscles is the *Thoma-Zeiss Hæmocytometer.*

This consists of (1) counting chamber ; (2) specially ground cover-glass ; (3) red corpuscle pipette ; (4) white corpuscle pipette.

The counting chamber is made of an outer flat ring of glass separated by a circular trench from a central platform, both being cemented to a glass slide. The outer ring is $\frac{1}{10}$ mm. higher than the platform, so that the cover-glass resting on it is separated from the platform by $\frac{1}{10}$ mm. The centre of the platform is ruled into minute squares with sides $\frac{1}{20}$ mm. Thus the area seen on looking down over each small square is

$$\frac{1}{20} \times \frac{1}{20} \times \frac{1}{10} \text{ c.mm.} = \frac{1}{4000} \text{ c.mm.}$$

As a rule the small squares are enclosed in sets of sixteen by double rulings, and each set of sixteen " small squares " is called a " large square."

The red corpuscle pipette is a capillary tube, graduated in tenths up to a mark 1, leading to a bulb which contains one hundred times the volume of the tube, and having above it the mark 101. The bulb contains a glass bead to facilitate mixing the blood with the Hayem's Fluid.

The white corpuscle pipette is similar but the bulb only contains ten times the volume of the tube, the marks being 1 and 11. Its use will be considered in dealing with the leucocyte count.

Procedure.—Draw up the fresh blood in the pipette to the mark 1, quickly wipe the tip, and draw up Hayem's Fluid to the mark 101. The bulb now contains blood diluted 1 in 100. The upper mark is 101, and not 100, because the stem of the pipette now contains one volume of Hayem's Fluid which is later discarded. The pipette is rolled round gently, allowing the glass bead to mix the blood uniformly. Next, after blowing away the pure Hayem's Fluid in the stem, a small drop of diluted blood is blown on to the ruled platform and the cover-glass lowered gently to avoid bubbles. The drop should be of such size that it does not run over the edge of the platform, and is most easily gauged by squeezing the rubber mouthpiece instead of blowing. The droplet is now examined on a level stage with the $\frac{1}{6}$-inch objective of a microscope, counting the number of corpuscles in each of the sixteen squares of a large square and drawing an average from the counts of at least five or six sets. As some corpuscles overlap the lines, it is advisable to include in any square those which overlap, say, the top and right-hand side only, those which overlap the other two sides being counted with other squares.

Computation.—Each square represents $\frac{1}{4000}$ c.mm. and the blood is diluted $\frac{1}{100}$.

Thus the number of red corpuscles per cubic millimetre is : 4000 × 100 × average number of corpuscles per square. The number computed is of course not absolutely accurate, but the margin of error is comparatively negligible.

A quicker way is to dilute the blood 1-200, in which case the total number of corpuscles counted in five " big squares " followed by 0000 is the number per cubic millimetre of blood.

Counting the Leucocytes

The second pipette is used, diluting $\frac{1}{10}$, and a different diluting fluid is used—Thoma's Fluid :

Acetic acid (B.P.) 1 c.c.
Sat. sol. Gentian Violet ·5 c.c.
Distilled water 100 c.c.

With this fluid the red corpuscles are laked and the leucocytes are slightly stained to make them prominent. In this instance only " large squares " are counted, as there are comparatively few leucocytes as compared to reds.

Computation.—Each large square is $\frac{1}{5} \times \frac{1}{5} \times \frac{1}{10}$ c.mm. $= \frac{1}{250}$ c.mm. and the blood is diluted $\frac{1}{10}$. Thus the number of leucocytes per cubic millimetre of blood is 250 × 10 × average number per *large* square.

Cleaning the Apparatus.—The pipettes are washed out with water, then alcohol, and finally ether, but the counting slide must only be washed with water ; alcohol or ether would dissolve the cement, and the ring and platform would come unstuck.

Estimation of Hæmoglobin

The best apparatus is the Gower-Haldane Hæmoglobinometer, being a combination of that of Gower and that of Haldane.

In Gower's apparatus a standard tube contains a solution of carmine identical in colour with 1 per cent blood in water. The blood to be examined is taken in a marked pipette and then expelled into a tube standing beside the standard one and graduated in hundredths. Water is added gradually and the blood mixed continually until the solution is the same as the standard. The graduation on the tube is now read off, and gives the percentage of hæmoglobin.

Haldane's method is very similar, but the measured quantity of blood is first laked with about ten volumes of water, and coal gas allowed to flow into the tube. This converts the hæmoglobin to carboxyhæmoglobin, and it is compared when diluted to a standard 1 per cent solution of actual carboxyhæmoglobin. This method has the advantage over that of Gower in that the two solutions to be compared are of the same substance ; but one must have a supply of coal gas laid on, and this is not always available in country districts.

Tallqvist's Hæmoglobin Scale may be employed. This consists of a booklet of absorbent papers. A drop of blood is touched with one of the strips and allowed to diffuse. It is then compared, before quite dry, with a standard scale of tints supplied in the booklet. With this quite simple method the error should not be more than 5 per cent.

Estimation of hæmoglobin is as necessary as counting the red corpuscles. For instance in chlorosis the number of corpuscles may be very nearly normal, but they contain little hæmoglobin. Conversely in pernicious anæmia the corpuscles are greatly reduced, but have a high hæmoglobin content. After severe hæmorrhage the reds are reduced, but have a normal hæmoglobin content.

One expresses the figures by employing the "Colour Index," which is

$$\frac{\text{Percentage of Hæmoglobin}}{\text{Percentage of Corpuscles}}.$$

	Per cent of Red Corpuscles.	Per cent of Hæmoglobin.	Colour Index.
Normal animal . . .	100	100	1
Anæmia from hæmorrhage	80	80	1
Chlorosis	80	40	$\frac{40}{80}={\cdot}5$
Pernicious anæmia . .	20	30	$\frac{30}{20}=1{\cdot}5$

Histological Examination

Blood may be examined fresh—a drop of blood between slide and coverglass—or fixed and stained in the form of a smear.

To make smears one may put a drop of blood on a slide and spread by touching the drop with the ungummed edge of a cigarette paper and then drawing the paper along. A better way is to put the drop nearer one end of the slide, and then, laying the edge of another slide through the middle of the drop, allow the drop to spread out to the sides of the slide and push or draw the second one along.

Fixing and Staining.—In many stains a fixative is included, making a preliminary fixation unnecessary.

Separate fixation may be accomplished by immersing the slides in bottles of various fixing reagents, *e.g.*

1. Absolute alcohol 2. Equal parts alcohol and ether	} 1 hour.
3. Formalin, 1 per cent.	1 minute.

Many stains are used for blood, and they vary in usefulness for different purposes. All however act on the same principle. When old, or alkaline, or specially treated solutions of methylene blue are left in contact with eosin, a chemical combination takes place, analogous to an acid combining with a base, and the resulting compound is eosinate of methylene blue.

Jenner's, Wright's, and Leishman's stains combine the processes of fixing and staining. For Giemsa's stain a previously fixed smear is required.

Jenner's Stain.—Immerse the unfixed smear in the stain for three minutes, wash in *distilled* water until it becomes bright pink, dry rapidly by blowing on the smear, and examine direct with $\frac{1}{12}$-inch (oil immersion) objective.

Wright's and Leishman's Stains.—Pour on a counted number of drops of the stain and leave for one minute. Add an equal number of drops of *distilled* water and leave for five minutes. Wash the smear until it turns pink, dry rapidly, and examine.

Giemsa's Stain.—The stain is used, freshly diluted, in the proportion of 1 drop of stain to 1 c.c. of distilled water. The smear must be previously fixed, and the stain is allowed to act for about fifteen minutes.

With all the above stains the basophile elements of the blood take the basic portion of the stain—the blue—and the acidophile elements take the acid portion and stain red.

Basophile elements are: Nuclei of leucocytes, and of red corpuscles if present, and basophile granules of leucocytes (mast cells).

Acidophile elements are: Red corpuscles, granules of polynuclear and eosinophile leucocytes.

The cytoplasm of the leucocytes stains very faintly blue or not at all, and the granules of eosinophile leucocytes stain more richly red than either the granules of the polynuclears or than the red corpuscles.

In examining stained blood films it is essential to use a $\frac{1}{12}$-inch (oil immersion) objective.

Morphology of the Blood Corpuscles

Red Corpuscles (Erythrocytes).—Normally these have the shape of a disc, concave on both surfaces. In the camel, however, they are oval. Mammalian red corpuscles are normally non-nucleated, but in birds a nucleus is present and the corpuscles are oval in shape. Their size, and number per cubic millimetre, vary in different animals. In stained preparations, red corpuscles stain pink, and rather more deeply at the periphery owing to greater thickness.

Abnormal Forms.—Any variation from the above description denotes an abnormality, except in some cases where the corpuscles appear crenated. This is due to faulty preparation of the film.

When normal in shape, but abnormally small, the corpuscle is known as a *microcyte*, and if too large as a *macrocyte* or *megalocyte*. When varieties in shape occur, such as oval, pear-shaped, fiddle-shaped, they are called *poikilocytes* and the condition spoken of as poikilocytosis. Nucleated red corpuscles are called *erythroblasts*, and if smaller or larger than a normal corpuscle are called *microblasts* and *megaloblasts* respectively. In some conditions also staining is not regularly pink, and a blue coloration is noticed. This may be in the form of a diffuse invasion—*polychromasia* or *polychromatophilia*—or of small well-defined blue points—*punctate basophilia* or *punctate degeneration*.

The importance of these abnormalities of red corpuscles is that they indicate disease—usually anæmia—although erythroblasts are not rare in very young animals, being probably a persistence from the fœtal state, in which the normal red corpuscles are nucleated. The observation and estimation as to increase or decrease of these abnormalities are of great aid in the prognosis of an anæmia from whatever cause.

Leucocytes.—There are several kinds of normal leucocytes, which are present in more or less constant proportions, varying of course in different animal species. The only way to find the proportion accurately is to count some hundreds of leucocytes and note how many of each kind are seen. In some diseases, variation from the normal is so pronounced as to make this tiresome procedure unnecessary. Apart from variation in the normal leucocytes, there are conditions in which new ones appear. These will be described after the normal ones.

Normal Leucocytes.—There are five varieties :

1. *Small Mononuclears* (Lymphocytes).—In size these are very little larger than the reds, but slightly variable. The nucleus occupies most of the cell and stains deep blue. The cytoplasm shows as a narrow area of very lightly stained blue around the nucleus.

2. *Large Mononuclears* (Hyaline leucocytes).—These are roughly twice the size of the small mononuclears and stain similarly. The nucleus does not form so large a proportion of the cell and is not usually spherical, being crescentic or kidney-shaped.

Intermediate stages are sometimes noticed between the large and small mononuclears.

3. *Polymorphonuclears* (Polynuclears).—Not quite so large as the large mononuclears. The nucleus is not spherical but variously lobed or twisted. The name of the cell was derived from this characteristic of the nucleus, which may be S, Z or U shaped. This nucleus stains of a bright lilac tint. The cytoplasm is abundant and contains many fine acidophile granules. These stain very faintly pink in a cytoplasm which stains either very faintly blue or not at all.

In the fowl, however, the picture is very different. The nucleus does not stain so deeply, and the granules are cigar- or spindle-shaped and stain a very deep red. Both in mammals and birds the polynuclear leucocytes are capable of amœboid movements.

4. *Eosinophiles.*—About the same size or slightly larger than the polynuclears, with a similar nucleus. In the cytoplasm are many very coarse, deeply red-staining granules, easily distinguishable from those of the polynuclear by their much greater size and deeper staining. Even in the fowl the eosinophile is easily distinguishable as the granules are round, whereas those of the polynuclears are spindle-shaped.

5. *Mast Cells* (Basophile leucocytes).—This is the rarest variety in all animals. They are little larger than the small mononuclears, which they closely resemble. The nucleus, however, does not stain so deeply, and the cytoplasm contains several basophile (blue-staining) or neutrophile (purple-staining) granules.

Abnormal Leucocytes.—In severe anæmias one may see immature forms of normal leucocytes, but there are also to be seen two definite forms of foreign leucocyte.

1. *Basophile Myelocyte.*—Large cells—even larger sometimes than the large mononuclears—with a roughly spherical nucleus. Both nuclei and protoplasm stain palely and the latter is granular, making the myelocyte distinguishable from the large mononuclear in which the protoplasm is hyaline.

2. *Eosinophile Myelocyte.*—Similar to the last, but the granules in the protoplasm stain red. They are easily distinguished from the normal eosinophile in having a round nucleus.

Blood Plates.—These are organized elements found in normal blood, being flat and about one-third the diameter of red corpuscles. They are very difficult to detect, and unless special methods be employed are not seen in smears. In the fowl, however, there are nucleated oval cells homologous with the mammalian blood plates. These are not quite so long, and about half the breadth of the red corpuscles. With blood stains, the nucleus is seen to be blue and the cytoplasm a paler blue. They are known as *thrombocytes*.

On the following page is a table of the *approximate average* constitution of the blood as regards the red and white corpuscles in various animals. As the variations of the normal are so wide it necessarily follows that the figures given are only approximately accurate.

Variation in Numbers of Blood Elements

1. *Variations in the number of Red Corpuscles.*—Increase in the number is known as *Polycythæmia*, and decrease as *Oligocythæmia* or *Anæmia*. There are many physiological causes of variation in blood count. For instance,

Animal.	Red Corpuscles.		Leucocytes.					
	No. per c.mm. (in millions).	Size (in microns).	No. per c.mm.	Percentages of Varieties.				
				Small Mono-nuclear.	Large Mono-nuclear.	Polymorph.	Eosino-philes.	Mast Cells.
Horse	7½-8	5½	8000	30	6	59	4	1
Ox	6-7	5½-6	5000-10,000	54	1·5	30	14	·5
Sheep	8	4½-5	8000	53	8	37	1·7	·3
Goat	10	4	10,000	not yet determined				
Dog	6	6-7	8000	20	6	68	6	very few
Cat	6½	6-6½	13,000	33	4	54	9	very few
Pig	8	5-6	20,000	44	3	50	2	1
Rabbit	6	6-7	11,000	50	2	45	1	2
Fowl	3	12 × 7	12,000-25,000	42	12	30	12	3

younger animals have a higher count than adults; males have a higher count than females or neuters; animals living at high altitudes have a higher count than normal average.

Administration of purgatives concentrates the blood by simple extraction of water.

Pathologically *polycythæmia* may be due to profuse diarrhœa, vomition or exudation; in the last, due perhaps to pneumonia or ascites. Lack of sufficient drinking water will produce the same effect.

Anæmia is due pathologically to many factors—hæmorrhage, malnutrition, loss of activity of blood-forming organs, and destruction of red corpuscles being the commonest. Hæmorrhage and malnutrition need no further discussion. Loss of activity of blood-forming organs may be due to specific diseases, especially when accompanied by toxin formation. Poisoning by lead, mercury or arsenic will produce the same effect by lessening blood formation.

Destruction of the red corpuscles is a feature of many pathological processes, mainly septic invasions, septicæmias, pneumonia, tuberculosis, equine hæmoglobinuria (azoturia), and many protozoan diseases (various redwaters).

Decrease in number of the red corpuscles to any degree is always accompanied by alteration in the appearance of the persisting corpuscles. According to the severity one observes variation in size, poikilocytosis, polychromasia, punctate degeneration, nucleated red corpuscles, and eventually introduction of myelocytes into the circulation.

2. *Variation in the Number of Leucocytes.*—If the number is increased this condition is *leucocytosis*, and if diminished, *leucopenia*. Physiological variations are common and follow various stages of digestion, parturition, and violent exercise. Very young animals constantly show a leucocytosis. Leucocytosis does not necessarily involve an increase in the number of all the varieties of leucocytes, and similarly with leucopenia.

(*a*) Leucocytosis.—Digestion in ruminants and herbivora generally causes a very small rise in numbers; if noticeable the polynuclears increase. Carnivora, however, show a large increase in small mononuclears (*lymphocytosis*) some hours after a big meal. One should bear this in mind when attempting a normal count, or the result may be misleading. New-born animals have a high number of leucocytes, especially of lymphocytes, returning gradually to the normal in a few weeks as a rule. Violent exercise induces a polynuclear leucocytosis.

Pathologically any acute inflammation causes leucocytosis, especially suppurative processes. In this case the polynuclears are mainly increased, and the more vigorous the resistance of the animal to the infection, the greater the leucocytosis. The common veterinary leucocytosis accompanies strangles, pleurisy, pneumonia (of all kinds), quittors, abscesses, etc., and tetanus.

Experimentally, leucocytosis is induced by introduction into the system of various chemical or bacterial products, the former class producing usually a transient effect. Camphor, quinine, pilocarpin, nuclein, strychnine, ethereal oils, and many metallic salts will cause leucocytosis. Hæmorrhage also is followed at an interval of several hours to a few days by increase in the number of leucocytes, being roughly in proportion to the amount of blood lost and to the recuperative power of the patient. It is thus a good guide to prognosis in cases of severe hæmorrhage.

The main individual leucocytoses and their causes are as follows:

Lymphocytosis.—Normally this occurs more in the young than in adults. Pathologically, lymphocytosis is most commonly seen in trypanosomiasis.

Eosinophilia (increase in eosinophiles).—The greatest and most common cause is *helminthiasis*. Almost any worm infestation may be responsible—ascarides, tapeworms or sclerostomes; markedly so in the case of the last named. Skin diseases may also be a cause, in direct proportion to the actual irritation caused, rather than to the extent of skin invaded. Thus in mange one would expect a greater degree of eosinophilia than in eczema.

Eosinophiles are diminished in malignant organic diseases, being almost absent immediately before death.

Polynuclear Leucocytosis.—Noticed in cases of pneumonia and all septic infections, *e.g.* fistulous withers, quittor, strangles, empyema. As the condition is constant in septic infections, it may aid in the diagnosis of deep-seated pus.

Hyaline Leucocytosis.—This is not commonly noticed in this country. Some degree of it accompanies protozoan diseases, *e.g.* redwater.

Mast Cell Leucocytosis is very rare and may be seen in leukæmia.

Myelocytes.—These foreign cells are seen in leukæmia and advanced anæmia. They are also occasionally seen accompanying polynuclear leucocytosis.

(*b*) LEUCOPENIA (decrease in leucocytes).—As in leucocytosis, not all varieties are equally affected. It is usually only noticeable following destruction of tissues responsible for leucocyte formation—as in tuberculosis or some other toxic diseases. Malnutrition and general debility other than debility due to worms may cause it. The condition may be induced experimentally with drugs—ergot, tannin, atropin.

Special Diseases affecting the Blood

Pernicious Anæmia.—This disease produces the most striking changes in the blood elements of any blood disease. It is entirely different from the homonymous human disease, having more the characters of a severe secondary anæmia. The red corpuscles are reduced, showing microcytes and megalocytes. Poikilocytosis, punctate degeneration and polychromasia, with presence of nucleated red corpuscles, are all to be seen. Leucocytes are also reduced, but not so much as the reds ; giving a spurious impression of leucocytosis.

The cause of the disease is usually obscure in animals and few cases are ordinarily seen.

Leukæmia (Leucocythæmia).—A primary disease of the blood-forming organs, and very rarely seen in the lower animals, possibly owing to the infrequency of blood examination. Two forms are recognized, viz. *Mixed-celled* or *Myeloid Leukæmia* and *Lymphatic Leukæmia.* In both forms there is great increase in the number of leucocytes and decrease in red corpuscles. In the former a great proportion of the new leucocytes are myelocytes, whereas in the latter they are mainly lymphocytes—even over 90 per cent of the total.

In both forms the spleen, lymphatic glands, and bone marrow show hyperplasia, but these structures are not softened, being either as firm as, or firmer than normal owing to simultaneous corresponding increase in connective tissue.

Throughout the body, in capillaries and lymph spaces, excess of leucocytes is observed, the variety seen being dependent on the form of leukæmia present.

Lymphatic leukæmia is the only form so far observed in the lower animals.

Pseudoleukæmia (Hodgkin's Disease).—Also sometimes called *Lymphoma* or *Lymphadenoma.* The main anatomical features are exactly similar to those in true leukæmia, viz. enlarged lymphatic glands, spleen and bone marrow, but it is easily distinguished in that the proportion of leucocytes in the blood is approximately normal. Of the domesticated animals the dog is the only one in which this condition is not rare.

General and Infectious Diseases affecting the Blood

Changes observed in the blood accompanying various diseases vary with the class of disease. For example, in septic inflammations, pneumonia, and tetanus there is leucocytosis, mainly polynuclear ; in anthrax or fowl cholera the specific organisms themselves are to be found in the circulating blood ; in glanders and contagious abortion, specific agglutinins are present in solution in the plasma, and proof of their presence is utilized in diagnosis. Again, helminthiasis will produce eosinophilia, and mononuclear leucocytosis is common in protozoan invasions.

Strangles.—In the early febrile stages the blood is concentrated owing to dehydration. Later polynuclear leucocytes are increased. When the abscess ruptures or is incised the leucocytes return to normal.

Equine Pneumonia.—Very similar blood features to strangles. There is primary concentration of the blood owing to the fever, giving the impression of polycythæmia. Later leucocytosis is seen, mainly polynuclear, and the number of red corpuscles becomes subnormal. The occurrence of leucocytosis is a favourable sign, but if the initial polycythæmia persists to the exclusion of leucocytosis, prognosis is very unfavourable, indicating in all probability necrosis or gangrene.

Fowl Cholera.—In naturally occurring cases, the blood symptoms are those of anæmia with moderate leucocytosis. Blood from a bird dead of the disease will show the causal organisms—small bipolar-staining bacilli.

Anthrax.—In cattle and sheep the blood is extensively invaded by the bacilli a short time before death ; so constantly, in fact, that if they are not to be found at the time of death in a suspicious case, one may be certain that anthrax is not the cause of death. In horses the organisms are present in the vast majority of cases, but in pigs death often occurs before the blood stream is invaded. The changes in the blood itself are very small. There may be a slight leucocytosis and anæmia following disintegration of red corpuscles. The most important features are the dark colour and tarry consistency, and the fact that it does not clot.

Microscopical examination is limited to a search for anthrax bacilli. Any of the Romanowsky stains are suitable, but the best method is M'Fadyean's. This method has the advantage of using a thick smear and is performed as follows: A thick smear is air-dried by waving the slide rapidly, and then incompletely fixed by heat, the slide being heated in a flame until only just uncomfortably hot to the back of the hand. The smear is now stained with 1 per cent methylene blue for a few seconds, washed thoroughly, and dried by blotting. When examined under the $\frac{1}{12}$-inch objective the bacilli are stained blue with a pinkish capsule, or if the capsules are broken up the material appears as pinkish amorphous masses. This pink reaction of the capsular material is even visible to the naked eye, for when the stained smear is held obliquely to the light it is seen to be a distinctly reddish blue, quite different from normal blood.

Glanders.—In the early stages of acute glanders there is a concentration of the blood, but later a moderate anæmia, accompanied by polynuclear leucocytosis. This latter becomes more pronounced when the disease assumes clinical proportions. In non-clinical cases the leucocytosis is said to be rendered more pronounced within twenty-four hours by the administration of mallein. Glanders is one of the diseases in which an appreciable amount of specific agglutinin is found in the plasma, and this is constantly used in some countries in diagnosing the disease—agglutination test.

Tuberculosis.—The blood changes in obscure cases are almost negligible; in fact within the bounds of normal variation. In advanced chronic cases there is said to be anæmia, but this is often unrecognizable as the blood is concentrated. Some investigators claim to get positive agglutination results with the serum, whereas others have completely failed.

Specific (or Contagious) Abortion.—Mares, cows, and ewes are affected, the common form being induced in each case by a specific organism (mare, *Bacillus abortivo-equinus*; cow, *Bacillus abortus*, Bang; sheep, a vibrio or spirillum). The only important change is the formation of a specific agglutinin in the plasma, but there is no visible change in the blood. One may diagnose the specific infection by drawing off some blood in a small sterile bottle, centrifuging after clotting, and testing the agglutinating power of the serum on emulsions of the specific organisms.

Protozoan Diseases

Texas Fever.—A piroplasmosis due to *Piroplasma* (or *Babesia*) *bigeminum* in cattle. In the acute stages the causal parasites are seen in the blood in the form of pale pear-shaped bodies, usually in pairs inside the red corpuscles. With the usual blood stains the cytoplasm of the parasites is pale blue, with a red chromatin body, near the pointed end. Irregular forms are seen, but in typical cases the two pointed ends of two parasites are close together with the bodies lying at a very acute angle to one another, being often almost parallel.

Usually only a small proportion of the corpuscles is invaded—often less than 2 per cent, but in fatal cases up to 10 per cent may be affected before death.

The changes in the blood elements are the result of invasion by the piroplasms. A large number of red corpuscles is destroyed, the count being reduced often to 2,000,000. Accompanying this destruction the signs of anæmia appear—nucleated reds, poikilocytes, polychromasia, etc. Leucocytes are increased, particularly the lymphocytes, and after recovery the number is said to remain higher than normal.

British Redwater.—Similar as regards blood changes to Texas fever, but the causal piroplasm is *Babesia divergens* (M'Fadyean and Stockman), so-called because two parasites in a red corpuscle lie at a very obtuse (divergent) angle to one another, often being almost end to end.

Canine Piroplasmosis (Malignant Jaundice of Dogs).—Caused by *Babesia canis* and is a very widespread disease.

The parasite is very like *B. bigeminum*, and further resembles it in that only a very small proportion of corpuscles is invaded. A striking point, however, is that, as the disease progresses, although the percentage of invaded corpuscles is low, any single corpuscle may contain a large number of parasites—12 or 16. In chronic cases so few corpuscles are invaded that there is great difficulty in finding them.

The blood changes in acute cases are similar to those in Texas fever—a progressive, severe anæmia, accompanied by leucocytosis; but in this case the increase is mainly in the polynuclears (Nocard).

Biliary Fever in Horses (*Equine piroplasmosis*).—This disease has two causal organisms—*Babesia caballi* and *Nuttallia equi*. Clinically the two are said to differ slightly. In the acute febrile stages the parasites are found in the red corpuscles. *B. caballi* has a morphology and distribution singly or in pairs similar to *B. bigeminum*. *N. equi* is a more slender parasite, and its most typical form is just after division, which is into tetrads, so that the corpuscle shows four parasites all at right angles to one another like a " Maltese cross " (Nuttall).

A larger number of corpuscles is usually affected than in Texas fever or malignant jaundice—up to 10 per cent or more in severe cases. Administration of quinine lowers the Maltese cross forms of *N. equi* by inhibiting division (Theiler).

The blood changes are exactly parallel to those in Texas fever.

Sheep Piroplasmosis (*Carceag*), due to *Babesia ovis* and common in Eastern Europe. The morphology of the parasite and the blood changes are very similar to those in Texas fever.

Babesia mutans.—Described by Theiler, occurring in South Africa, often in company with other parasites. The parasite itself is fairly harmless, but is important in that it may be mistaken for a stage of *Theileria parva*, the causal parasite of East Coast fever.

B. mutans is smaller and more rod-shaped than *B. bigeminum*.

East Coast Fever (Rhodesian Tick Fever).—The causal parasite is *Theileria parva*. Most of the life-cycle of this parasite is passed outside the blood stream—mainly in the lymphatic system. However, *towards the end* of the course of the attack, sexual forms of the parasite appear in the red corpuscles. They are rather small and rod-shaped. A larger number of red corpuscles is invaded as time progresses. During the course of the disease the parasite is not present in the blood, but there is a great increase in mononuclear leucocytes.

Trypanosomiasis.—Any disease caused by a *trypanosome* is so named. All trypanosomes are a common general structure, varying with the individual species. There is a fusiform body with a large nucleus and a second chromatin body called the *centrosome* or *blepharoplast*. Along one side of the body is a filmy *undulating membrane*. From the centrosome rises a *flagellum* which runs along the free edge of the undulating membrane and is usually continued beyond its limit. The anterior end is the one towards which the flagellum runs, since the organism moves with this end foremost. Multiplication is asexual by vertical fission. Most trypanosomes are 20-30 μ long.

As it is very difficult to distinguish the various trypanosomiases clinically, so is it to recognize the individuals in the blood. However, the combination of clinical symptoms and a blood examination renders diagnosis more feasible. The blood symptoms are similar in all cases. There is a progressive diminution in reds, low hæmoglobin content, and mononuclear leucocytosis. Towards the end (death) of the disease myelocytes appear in the blood.

Examination for presence of trypanosomes may be made with a drop of fresh blood between slide and cover-glass. In these circumstances one may see the moving trypanosomes or notice that the red corpuscles are being agitated by some body. To attempt to identify a trypanosome individually a stained smear is necessary.

Trypanosomes are spread from animal to animal by parasites—probably always by biting flies, in whose digestive tract the trypanosome sometimes undergoes a certain development. There is one most important exception, viz. *Dourine* or *Mal de Coït*, which is spread only by copulation.

The table below shows the most common trypanosomiases.

Disease.	Causal Trypanosome.	Discovered by	Distribution.	Animals Affected.
Surra . . .	T. evansi (Steel, 1885)	Evans, 1880	Asia generally and N. Africa	Equidæ Camel Dog Elephant
Nagana . .	T. brucei (Plimmer and Bradford, 1899)	Bruce, 1894	Africa generally	Most mammals
Mal de Caderas .	T. equinum (Voges, 1901)	Elmassian, 1901	S. America	Horses Dogs
Dourine . .	T. equiperdum (Doflein, 1901)	Rouget, 1894	S. Europe W. Asia N. Africa N. America	Equidæ

Diseases of Indefinite Origin.—These diseases are mainly due to ultra-visible viruses, *e.g.* rabies, rinderpest, swine fever, and equine pernicious or infectious anæmia.

The virus although invisible and filtrable may be present in the blood; which can be proved in positive cases by animal inoculation with filtered blood.

With rabies the leucocytes, mainly the polynuclears, increase constantly up to the point of death. In rinderpest there is an initial increase, then a decrease, and finally another increase. In swine fever and equine pernicious or infectious anæmia, on the other hand, there is a steady decrease both in leucocytes and red corpuscles, and in the latter disease the ordinary blood signs of anæmia.

Helminthiasis.—The blood may afford useful accessory evidence in aid of diagnosis. In all forms of helminthiasis, but chiefly in sclerostomiasis, there is a pronounced eosinophilia so that cases of colic of obscure origin may be diagnosed as resulting from worm infection. Acute indigestion or impaction does not materially affect the leucocytes, and in enteritis there is polynuclear leucocytosis.

In some cases of filariasis, as in the case of *Filaria immitis* in dogs, the worm embryos themselves may also be present in the blood.

Acariasis (Mange).—If severe, acariasis will induce eosinophilia, particularly in dogs and cats. It appears that the degree of eosinophilia is in direct relation not to the extent of infestation but to the amount of actual irritation or severity of dermatitis. Sarcoptic mange is the best example, and this of course has a simpler method of diagnosis.

J. T. E. and S. C. J. B.

DISEASES OF THE RESPIRATORY APPARATUS

Diseases of the Nasal Cavity

Nasal Catarrh; Coryza; Rhinitis.—This affection is a catarrhal inflammation of the nasal mucous membrane.

Causes.—In some instances nasal catarrh is a primary affection, while in other cases it is symptomatic of general catarrhal affections as strangles, influenza, and pneumonia in the horse, and distemper in the dog. In primary nasal catarrh the common cause is bacterial, and in such cases the condition must be regarded as infectious. Nasal catarrh may also be caused by other irritants in the nasal cavity, such as dust, pollen (hay fever), foreign bodies such as an ear of wheat (Nunn), irritating vapour, such as ammonia, chlorine, iodine, or sulphurous acid gas (fumes of burning sulphur). Nasal catarrh may also arise from the presence of parasites, larvæ of *Œstrus ovis* in sheep and *Pentastomum tenioides* in dogs.

Sudden changes of temperature and exposure to inclement conditions are sometimes regarded as responsible for nasal catarrh. Such conditions may predispose to nasal catarrh when the almost ubiquitous organisms are present, but it is difficult to understand how exposure to cold could of itself set up inflammation of the nasal mucous membrane.

Acute and chronic nasal catarrh are recognized.

Acute Nasal Catarrh.—*Symptoms.*—The chief symptoms are those of local irritation of the nasal chamber, such as snorting in the larger animals and sneezing in the smaller ones. This is soon followed by a discharge from both nostrils which at first is thin and watery, but soon becomes thick and tenacious and turbid. In some cases it may become creamy owing to the presence of large numbers of epithelial cells and leucocytes. It may cake round the margins of the nostrils, forming crusts, in the dog frequently producing cracking and great soreness of the muzzle. The mucous membrane is injected. The lachrymal *ductus ad nasum* may become occluded, resulting in an overflow of tears, suffusion of the eyelids, and sometimes associated with conjunctivitis. The sub-maxillary lymphatic glands may also be slightly enlarged and hot, particularly in the horse. The temperature may or may not be altered, depending on the severity of the attack. It is frequently, however, elevated by 2° to 3° F., but constitutional disturbance is rarely severe. When the nasal catarrh is excessive there may be difficulty in breathing, producing audible respiration almost amounting to roaring in horses and cattle, while in the dog and cat a sniffling sound is produced and oral respiration. The latter animals may also rub their faces with their paws, and not infrequently rub their noses along the ground or against any convenient object. In mild cases the affection runs through its acute stages in four or five days, and recovery may be complete in eight to ten days. In severe cases the catarrh may extend into the pharynx and larynx, or into the air sinuses, and set up a chronic nasal discharge.

Chronic Nasal Catarrh.—*Symptoms.* — This form is usually a sequel to an acute attack. There is a persistent or intermittent nasal discharge which may vary considerably in character. It may be glairy and tenacious, while in other cases it may be thick and creamy, and one or both nostrils may be affected. In some cases the discharge is blood-stained, and at other times when there is necrosis of the turbinated bone, or where the facial sinus has become diseased through bad teeth, it is fœtid in odour (ozæna). The mucous membrane often appears somewhat congested and streaky and dirty-looking. In rare cases ulcers may be found on the nasal mucous membrane, but in all such cases in horses glanders should be suspected and the animal tested accordingly.

Treatment.—All cases of acute nasal catarrh should be isolated, as they may be centres of infection. Horses must be taken from work, and all animals must be put under comfortable conditions as to warmth and general hygiene in well-ventilated surroundings. In cold weather clothing should be applied. Medicated inhalations may be resorted to. In the case of the larger animals this may be effected by placing a quantity of hay or chaff into a deep bucket, sprinkling on to it such agents as terebene, creosote or oil of eucalyptus, and then pouring boiling water over it and placing the bucket in a sack. The sack is then held over the animal's face, care being taken to exclude the eyes. Sometimes the medicament is dropped on to a hot brick and the fumes inhaled, but this method is not so good as the former since the water-vapour or steam is of itself soothing and also assists in reducing the consistence of the nasal discharge and facilitating its removal. In the smaller animals the steaming kettle may be used, or boiling water may be placed in a jug or basin with medicament added and held under the animal's head.

Irrigation of the nasal chamber may also be resorted to with a mild antiseptic, such as 5 per cent boracic acid solution, ½ per cent carbolic acid or tannic acid, or simple salt solution gr. iv.-v. to the ounce, and bicarbonate of soda in similar strength. Irrigation of the nasal chamber, however, in veterinary patients creates considerable distress and is not very easily carried out. It is doubtful if there is any compensating advantage compared with inhalation in simple nasal catarrh. If there is any tendency to soreness of the nostrils they should be anointed with boracic ointment, which should be gently but thoroughly rubbed in once or twice daily. This is more frequently called for in the dog than in other species. Only very simple measures are called for internally. In the case of the larger animals sulphate of magnesia ℥ii.-iv. once daily in the drinking-water generally suffices. Alternatively potassium nitrate ʒii.-iv. or solution of acetate of ammonium ℥iv.-v. may be administered in the drinking-water. In cases associated with fever antipyretics must be given such as quinine sulphate ʒi. or sodium salicylate ʒii.-ʒiv. These may be administered in bolus or mixed with treacle and applied to the back of the tongue. In dogs and cats syrupus scillæ m. xv.-ʒi. may be administered twice daily. If the animal is feeding it will probably take it voluntarily in a little milk. In febrile cases sulphate of quinine gr. i.-ii. or aceto-salicylic acid gr. ss.-iii. will be found very serviceable.

In chronic nasal discharge in horses (nasal gleet) the source of the discharge may be the facial sinuses as the result of empyema or of diseased teeth. In the latter case the discharge is usually very fetid. A chronic discharge may also be due to pus in the guttural pouch. These conditions call for special surgical measures.

Epistaxis ; Rhinorrhagia ; Hæmorrhage from the Nose.—Under this heading is included hæmorrhage from vessels in the nasal cavity. The condition is very frequently a surgical one, and is brought about by injuries in the nasal cavity and foreign bodies. It may also occur, however, in the course of various specific affections such as glanders and purpura hæmorrhagica in horses, rinderpest and malignant catarrh in cattle, and sometimes in distemper in the dog. Bleeding from the nose may also result from stings by various flies, or from the presence of parasites, such as the larvæ of the *Œstrus ovis* in sheep and the *Linguatula tœnioides* in the dog, and leeches in the horse. In rare cases bleeding from the nose may result from violent exertion, as in racehorses and hunters, while at other times it results from simple rupture of capillaries, particularly in fat animals, from unknown causes.

Symptoms.—Blood may be seen escaping from one or both nostrils and may be small in degree, merely a trickle, or it may be escaping in a well-marked stream. In medical cases it is usually capillary in origin. When it occurs in the course of a specific disease it is usually associated with catarrhal discharge which may be rendered diffusely blood-stained or only blood-streaked. The bleeding may cease spontaneously, but is frequently renewed on exertion. The condition may be distinguished from hæmorrhage in the lungs by the fact that in the latter case it is usually churned up into foam, and is accompanied by coughing and some dyspnœa.

Treatment.—Owing to the natural tendency for capillary hæmorrhage to cease spontaneously no drastic measures are called for in medical cases. Complete rest and freedom from exertion or excitement are necessary. If the bleeding should be persistent, however, the nasal chamber should be irrigated with a weak solution of some hæmostatic such as tincture of hydrastis (1-8), extract of witch-hazel (1-40), adrenalin 1 in 10,000-20,000, alum 5 per cent, or solution of perchloride of iron 5 per cent. Infusion of oak galls or of oak bark may also be used. In very persistent cases if the hæmorrhage is copious the hypodermic injection of ergotin may check the hæmorrhage. (For the treatment of surgical conditions, see Vol. II., p. 796).

Diseases of the Larynx

Laryngitis ; Inflammation of the Mucous Membrane of the Larynx ; Laryngeal Catarrh

Inflammation of the mucous membrane of the larynx is a very common condition, and in the majority of instances is due to organisms. Laryngitis may be primary in incidence, or it may occur as an extension from nasal catarrh or pharyngitis, or it may be secondary to inflammation of the trachea and bronchi. It often occurs in the course of specific affections such as influenza, strangles, purpura hæmorrhagica, and glanders in the horse; tuberculosis in cattle and swine; swine fever; and distemper in the dog. Foreign bodies in the larynx and faulty drenching with irritant substances may set up laryngitis. It may also be due to the inhalation of dust or irritant fumes or gases, while a peculiar form of œdematous laryngitis may be met with in the horse as the result of poisoning by *Lathyrus sativus*.

Acute and **chronic laryngitis** may occur, the latter form being frequently a sequel to the acute. Pathologically laryngitis may be either **catarrhal, ulcerative, croupous, diphtheritic** or **œdematous.**

Acute Laryngitis. — *Symptoms.* — The first symptom observed is usually a cough, harsh and dry at the commencement, but becoming moist and softer. Fits of coughing are also set up after feeding, but not so prominently as in the case of pharyngitis. A sudden change of temperature also sets up coughing as the transference from a cold into a warm atmosphere or *vice versa*. There is a slight watery nasal discharge which may become thicker and more copious as the case progresses. On manipulation of the larynx tenderness is evinced and coughing and gulping may be set up. In the horse and ox audible respiration often occurs, while in the dog there is frequently a hacking cough followed by a shaking of the head and spurious attempts at vomition, resulting only in the expulsion of a small amount of frothy mucus. The temperature in laryngitis is variable, but in the majority of instances is elevated two or three degrees. One of the great dangers to be feared in acute laryngitis is a sudden onset of œdema of the larynx, in which case the glottis may become almost completely closed, breathing becomes very distressed, roaring is set up, and unless the condition is speedily relieved death may follow from asphyxia.

The *course* of acute laryngitis may extend from a few days to a fortnight, and may terminate in complete recovery, or the condition may pass on to chronic laryngitis, while in the horse after apparent complete recovery roaring or whistling may remain as a sequel.

Chronic Laryngitis.—*Symptoms.* — There is usually the history of an acute attack of laryngitis. After the acute symptoms have passed away there remains a more or less persistent hard cough, often dry, sometimes moist. During and after the fits of coughing there is audible respiration. The larynx is left in a very irritable condition, and fits of coughing are very easily set up by slight exertion. Manipulation of the larynx does not reveal tenderness as in the acute form, and there is rarely any elevation of temperature. This form is very likely to be followed by whistling or roaring in the horse.

Treatment of Laryngitis.—It is well to practise isolation at once in cases of laryngitis, on account of the frequency with which the condition is contagious. The patient must be placed in comfortable and warm quarters with plenty of fresh air and free from dust, while dusty bedding and dusty foods must be avoided. The patient should be clothed, and in the case of the horse a hood and bandages may also be necessary. Relief may be afforded locally by the application of poultices or mild blisters or stimulating liniments or hot cloths to the throat. Medicated inhalations as recommended in nasal catarrh should also be resorted to. All pressure on the throat must be avoided, as of throat lashes or head-collars.

Internally drenches should be avoided wherever it is possible. Medicaments may be administered in the form of electuary, amongst the best for the larger animals being chlorate of potash, camphor, and extract of belladonna. Glyco-heroin ℥ss. or chlorodyne ʒii. administered in water ℥iii.-iv., and allowed to trickle slowly into the mouth, has often been found useful. In the smaller animals compound tincture of camphor mm. x.-xxx. in an equal quantity of glycerine affords considerable relief. Other useful agents are syrup or tincture of squills and ipecacuanha. In cases where the coughing is very persistent and distressing it may be necessary to administer a course of bromides to obtain relief. In chronic laryngitis, where the catarrh is very tenacious and difficult to expel, a course of potassium iodide or iodide of iron will often bring about good results, while at other times a course of arsenic such as Donovan's solution will be found helpful.

Croupous Laryngitis; Diphtheritic Laryngitis.—In this form of laryngitis a diphtheritic deposit is produced on the laryngeal mucous membrane, often at the same time as the pharynx and trachea are similarly affected. It is, however, a rare affection in the domesticated animals, but is occasionally met with in the pig in the course of swine fever. It may also be produced in other animals by inhalation of irritating gases such as ammonia, chlorine, etc.

Symptoms.—These are similar to those of acute laryngitis, but there is usually greater interference with respiration both inspiratory and expiratory. It is difficult to distinguish in the early stages from acute laryngitis, but after several days shreds of the fibrinous deposit become detached and are expelled with the nasal discharge. In bad cases there is a danger of death from asphyxia.

Treatment.—This is on the same lines as recommended for acute laryngitis. In those cases where asphyxia threatens, tracheotomy should be performed.

Œdema of the Larynx; Œdematous Laryngitis

This condition may occur as a complication of acute laryngitis, or in the horse it may be set up as the result of poisoning by *Lathyrus sativus* (p. 516). It gives rise to more respiratory distress than any other form. The glottis may be almost completely closed owing to the œdema. Breathing becomes very difficult, roaring is set up and dysphagia, food and water returning from the nostrils. Unless it is speedily relieved asphyxia may occur.

Treatment.—Owing to the danger of rapid asphyxiation one must be prepared to insert a tracheotomy tube at short notice. If it occurs in the course of acute laryngitis the same medicinal treatment should be persisted in, but potassium iodide is particularly indicated. When the condition occurs as the result of lathyrism, a purgative should be given and a complete change of food ordered.

Laryngismus Stridulus; Spasm of the Larynx; Spasm of the Glottis

This is a condition of spasm of the constrictors of the larynx, and has been met with in horses and dogs. The cause of the condition is unknown, but it is thought to be a neurosis, the symptoms being brought about reflexly by indigestion through the pneumogastric nerve. A similar condition may also be caused by increased irritability of the nerve terminals in the larynx and pharynx, and attacks are sometimes brought on by inhalation of irritant fumes or by the administration of hot liquids or those containing irritant substances. Hutyra and Marek suggest that it only occurs in horses that have had tracheotomy performed, but spasm of the larynx is certainly not limited to such horses. In the course of an attack the glottis is suddenly narrowed during inspiration, thus resulting in the production of an abnormal sound at the larynx and a variable degree of dyspnœa. The condition is likely to be recurrent, but it rarely has any seriously injurious effects.

Symptoms.—These are usually rapidly developed, and may come on when the animal is at rest or when at work. The duration of the attack is variable, it may last just a few minutes or for hours. There is a suddenly occurring inspiratory dyspnœa, and at the same time a sharp spasmodic crowing noise is produced at the larynx. These symptoms may appear very alarming to the owner but may pass off just as suddenly as they arose. It is probable that intermittent attacks of spasm of the larynx are responsible for some of the cases of intermittent roaring in the horse.

Treatment.—The attacks may be cut short or reduced by the inhalation of amyl nitrite or chloroform, and in other cases by the inhalation of ammonia. In recurrent or persistent cases potassium or ammonium bromide or chloral hydrate should be administered to allay the nervous reflex and to ward off the spasms. If these cannot be administered by the mouth, as in cases where the attacks are still in existence, they may be injected per rectum. In very exaggerated cases, if dyspnœa should threaten tracheotomy should be performed. After the acute attack has passed the patient should be treated for indigestion, which is frequently the cause of the condition. A laxative should first be administered, followed by a course of alimentary and nerve tonics including nux vomica, gentian, and arsenic. The diet should be light and easily digested, and light exercise should be prescribed. In those cases where there is excessive irritability of the pharyngeal and laryngeal mucous membranes, local sedatives may be administered, such as small doses of belladonna in electuary in the horse, or compound tincture of camphor and glycerine in the dog.

Laryngismus Paralyticus; Whistling or Roaring

Whistling and roaring are very common affections in the horse and consist of the production of abnormal sounds at the larynx during inspiration.

Etiology.—In the majority of cases the affection is due to paralysis of the left recurrent (inferior laryngeal) nerve associated with atrophy of the muscles supplied by it, namely, the *thyro-arytenoideus*, the *crico-arytenoideus posticus*, *crico-arytenoideus lateralis*, all of the left side of the larynx, together with the left portion of the *arytenoideus*. In some cases these muscles undergo fatty degeneration. Roaring may also be set up by thickening of the laryngeal mucous membrane as the result of chronic laryngitis, or it may occur during the course of œdematous laryngitis. In other cases tumours pressing on the larynx from the outside, or new growths or polypi, may be responsible for it, as may also calcification of the cartilages of the larynx. In these latter cases abnormal respiratory sounds are produced by forced exercise both during inspiration and expiration, thus differentiating the condition from that form due to paralysis of the left recurrent nerve. In cases of defective heart in the horse, particularly in debility or dilatation of the ventricles, similar inspiratory and expiratory roaring is frequently produced. The nervous form is, however, responsible in the British Isles for 90-95 per cent of cases of roaring, and in the majority of these cases, probably 90 per cent, the left side alone is affected. In the remaining cases both sides are affected. During the examination of the interior of the larynx in a large number of cases (some hundreds), the writer has never yet seen a case of right-sided paralysis to the exclusion of the left side. Hobday reports having seen one such case.

The causes of paralysis of the recurrent nerve are somewhat obscure. There can be no doubt that heredity is an important factor, and especially is this the case in long-necked horses. The effect of contagious diseases is also a matter of great importance, and it is quite certain that strangles and influenza are frequently responsible for the development of recurrent paralysis. It is difficult, however, to understand, if these diseases are primary causes, why it is that the left side alone is so frequently affected and the right side so rarely involved. It is quite conceivable that the hereditary factor is still the main feature, and that the infectious catarrh and the toxins produced only serve to aggravate the hereditary factor by debilitating and increasing the irritability of the nerve terminals in the areas affected by the disease. On the other hand, certain poisons have undoubtedly a special affinity for nerve tissues, as is shown in lathyrism and in lead poisoning, in both of which roaring may occur. These examples are often quoted in support of the toxic theory of causation, and are said to support a "diseased *vagus centre*" theory of origin. If, however, the condition were due to a brain lesion it is difficult to understand why both sides are not equally affected. The difference in the course of the left and right recurrent nerves may afford a clue to the exciting cause of roaring, the left recurrent nerve having the longer course, and passing round the aortic arch. In this way it becomes affected by any enlargement or slight alteration in position of the heart and aorta which would cause a stretching of the nerve and might ultimately interfere with its functions. Certainly, as already stated, a considerable number of cases of roarers are affected with enlargement or dilatation of the heart. It might, however, be argued that the latter is a result and not a cause of the roaring. As against this is the fact that numbers of roarers in the writer's experience are not affected with complete paralysis of either recurrent nerve, but are affected with cardiac debility and dilatation. In such cases operative treatment on the larynx is obviously unlikely to produce any permanent beneficial results, and the writer is convinced that this condition is responsible for a number of those cases which have been operated upon by excision of the laryngeal ventricle in which temporary improvement has been followed by recurrence of the roaring. In his view the temporary improvement was only the result of enforced rest, and some recovery in the condition of the heart, the defective condition recurring soon after the horse was again put to forced exercise.

Mechanism of Roaring.—During normal inspiration the arytenoid cartilages are drawn outwards towards the wall of the larynx, and by that action the vocal cords are approximated to the laryngeal wall, thus closing the ventricles, which are then virtually only potential sacs. In this way perfectly free ingress of air is permitted. During expiration the arytenoids and vocal cords are relaxed, and the ventricles become actual sacs. This causes practically no obstruction since the free edges are directed away from the direction of the air current. In this way, then, there is no impediment to the ingress or egress of air during normal respiration.

When there is paralysis of the left recurrent nerve the intrinsic muscles attached to the left side of the arytenoid are functionless or almost so, consequently the arytenoid and its corresponding vocal cord are not drawn aside during inspiration, and the left laryngeal ventricle remains open with the free margin somewhat loosely directed against the air current. During rest, or on slight exertion, this obstruction may not cause any appreciable inconvenience, as a sufficient supply of air is allowed without any increased action of the dilators of the larynx. During work or forced exercise, however, a much larger supply of air is needed and the force of the air-current is greatly increased. It is then that an open ventricle, such as results from paralysis of the dilators, becomes an obstruction. In such a case the ventricle of the paralysed side becomes distended with air, and the air rushing into it and out again round the now tense vocal cord produces the sound which, according to the pitch, we designate whistling or roaring. This corresponds somewhat with the sound produced by blowing across the neck of an uncorked empty bottle. Just as in this case there is no vibration of the rim of the bottle, so it is probable that there is little or no vibration of the vocal cord or arytenoid to produce the sound. It would also appear that the difference in the pitch of the sound is due to the degree of relaxation of the arytenoid and vocal cord. When the dilators of the larynx are only partially paralysed, the ventricle is partially closed during inspiration, and the opening into it being more slit-like, a higher pitched sound (whistling) is produced than when paralysis is complete, and there is no degree of approximation of the vocal cord to the wall of the larynx, in which case the deeper note (roaring) is produced. The degree of respiratory distress corresponds directly with the amount of obstruction during inspiration. This fact will also justify the view held by many practitioners that roaring is a much more serious condition than whistling.

Symptoms of Laryngismus Paralyticus.—The characteristic symptom of this condition consists in the production of an abnormal sound at the larynx during forced exercise. The sound is occasionally but rarely produced when at rest. When the horse is put to work either trotting or cantering, or in the case of a draught horse when pulling a load, particularly up an incline, a slight whistling sound may be heard during inspiration, which may become louder and deeper as the exercise proceeds. In other instances the sound is deeper and more sonorous from the onset. If the exertion is further persisted in, respiratory distress and dyspnœa are set up, the head being extended and the nostrils dilated, the chest wall and flanks working excessively. In some cases the horse may be unable to proceed further, and, if he is forced to do so, may actually fall. When the horse is brought to rest the noisy sound disappears and breathing may be restored to normal within a few minutes. In pronounced roarers there may be a cough which, although not diagnostic, is sufficiently characteristic to afford grounds for suspicion as to the existence of paralysis. It is a deep, hollow prolonged cough (sepulchral cough). Many practitioners attach considerable importance to the character of this cough, and systematically "cough" a horse for diagnostic purposes by compression of the larynx with the thumb and fingers. Similarly, grunting will often indicate a roarer—a long deep grunt emitted when the horse is placed against a wall with his head held short and is suddenly threatened with the stick (bulling). In this connection, however, it should be borne in mind that although a peculiar grunt may make one highly suspicious that the horse is affected with paralysis of the larynx, yet there are numerous examples of roarers that are not grunters and *vice versa*. In roarers there is frequently a slight watery nasal discharge, but in many cases this is absent. Neighing is also slightly altered and is said to be more hoarse than normal. On manipulation of the larynx from the outside, atrophy of the muscles may be appreciated, and the arytenoid cartilage of the left side is felt to be more freely movable than normal. Great delicacy of touch is needed for this manipulation, and although some veterinary surgeons assert that they can in this way definitely diagnose the condition, it is unwise to rely upon it to the exclusion of forced exercise and the actual production of the abnormal sounds.

The greatest difficulty, however, is encountered in the case of intermittent whistlers and roarers. It is a matter of common experience that a horse may be a whistler one day, and although submitted to the same or even more severe tests he may fail to reveal any unsoundness the following day. Such cases are frequently the cause of litigation. The intermittent character of nervous symptoms as affecting the various parts of the body is commonly recognized, and intermittent roaring is probably due to the same causes. It is more likely to be met with in the early stages of the disease than in well-established cases.

Prognosis.—There is practically no likelihood of established roarers ever becoming sound in wind. The condition is frequently progressive, and animals affected in a slight degree may gradually become so badly affected as to be rendered incapable of work. In other cases, however, the respiratory distress may be slight in degree, and a horse may be able to perform even considerable exertion without inconvenience. Some animals that are affected with laryngeal paralysis and whistling, after an illness such as strangles, occasionally recover when their general health and condition are restored.

Treatment.—It is very doubtful whether curative measures ever have much beneficial effect. In early cases, however, the subcutaneous injection of liquor strychnia over the left side of the larynx has been practised, and good results claimed for it, and the same applies to the internal administration of potassium iodide and arsenic. The treatment, however, is more likely to be palliative than curative.

Preventive Measures.—These may be adopted in the case of horses affected with catarrh of the upper air passages or such affections as strangles or influenza. The administration of potassium iodide in such cases will reduce enlarged glands and also act as an eliminative of toxins. Such animals should also have a long period of convalescence and should not be given any forced exercise until they have completely recovered from the primary affection.

Various *palliative measures* have been resorted to from time to time. These are mainly surgical and consist either of the insertion of a tracheotomy tube or of various operations on the larynx itself (see Vol. II.).

Diseases of the Trachea and Bronchi

Bronchitis and Tracheitis

Inflammation of the trachea and of the larger bronchial tubes frequently occur together. When the smaller bronchial tubes are affected the condition is more commonly called capillary bronchitis, and this gradually merges into broncho-pneumonia. It is, moreover, comparatively rare to find even the medium-sized bronchial tubes affected without the lung tissue being involved. There is usually present some form of broncho-pneumonia, and sometimes collapse of a portion of the lung tissue.

Causes of Bronchitis.—Bronchitis is always due to the presence of an irritant in the bronchial tubes. The common irritants causing bronchitis are as follows: (*a*) Bacteria, which may have gained access during respiration or from the lung. Bronchitis occurring in the course of various contagious diseases is due to this cause, as in the case of influenza in the horse, distemper in the dog, etc. (*b*) Animal parasites, particularly strongyles. These are the commonest cause of bronchitis in cattle and sheep. (*c*) Chemical and mechanical irritants, such as irritant gases that have been inhaled, or solids or liquids of an irritant character that may have passed into the trachea in faulty or careless drenching. In some diseases, especially those interfering with swallowing, such as tetanus, the passage of saliva into the trachea may be followed by bronchitis and pneumonia. (*d*) Exposure to cold and inclement conditions is often followed by bronchitis, especially if the animal is exhausted from any other cause. In such cases, however, bacteria can always be found in large numbers in the inflamed mucous membranes, and it is probable that they are the actual exciting causes of the inflammation, and that the exposure simply provided conditions which enabled the organisms to penetrate and set up inflammatory changes.

Anatomical Changes.—On wiping the discharge off the bronchial mucous membrane the latter is generally seen to be reddened either diffusely or in patches, and sometimes more or less thickened. This is particularly seen in the earlier stages, but is not so well marked in later stages. The most important feature is the copious exudate in the bronchi made up partly of inflammatory liquid and partly of mucus secreted in excessive quantity in the inflamed bronchi. The discharge in the bronchi is sometimes churned into a froth by the passage of air through it. When the exudate is accompanied by excessive desquamation or shedding of epithelial cells the condition is called **catarrhal bronchitis,** and the discharge is then more or less opaque. If the exudate contains large numbers of leucocytes so as to make the discharge almost like pus it is called **purulent bronchitis.** At other times the exudate contains a large amount of fibrin which coagulates inside the bronchial tubes, and in some instances actually forms a plug occluding the bronchus — **croupous bronchitis.** This form is most likely to be met with in cases due to strong chemical irritants. In other cases the exudate may be offensive in odour, and it is then called **putrid bronchitis**, and is due to the invasion of septic organisms. In **parasitic bronchitis**, as seen in the ox, sheep, and pig, the small threadworms can be seen in considerable numbers in the trachea and larger bronchi. In portions of the lung tissue there may be seen areas of emphysema, and in other places patches of collapse of lung tissue. The bronchial lymphatic glands may also show some inflammatory changes. They are frequently swollen, enlarged and juicy, and in rare cases hæmorrhagic.

Clinically, **acute** and **chronic bronchitis** are recognized.

Acute Bronchitis; Acute Bronchial Catarrh.—*Symptoms.*—The symptoms are mainly due to irritation of the bronchial tubes by the primary irritant and the inflammatory products, to the mechanical interference with oxygenation of the blood, and to the absorption of toxins. Respiratory symptoms are most pronounced in acute bronchial catarrh and usually commence with a cough which is harsh and dry at the commencement, becoming soft and moist as the condition advances. Any exercise often produces fits of coughing, as may be seen when a horse is trotted a few yards or when cattle and sheep with parasitic bronchitis are roused and made to run even a few steps. In the dog, spasms of coughing are frequently followed by attempts at vomition, with the ejection of white frothy mucus from the throat, but only very rarely accompanied by ingesta. There is a nasal discharge which at first is rather thin and watery, then becomes somewhat mucoid and sticky, later becoming much more copious, thick, and catarrhal. This discharge may continue for some days after the acute stages of the affection have passed, when the patient is well on the way to recovery. Respiration is blowing, and some degree of inspiratory dyspnœa is produced in extensive cases owing to the partial or complete occlusion of bronchial tubes. Bronchial sounds are often audible without auscultation. The mucous membranes have a dull slaty-bluish tint owing to the non-æration of the blood, and there is very frequently a catarrhal discharge from the eyes. The pulse is accelerated, soft and large. The temperature is usually elevated from two to three degrees, and the appetite is capricious. In cattle the muzzle becomes dry, rumination is partially suspended, and lactation of milch cows markedly diminished. In dogs there is frequently a caking of discharge about the nostrils, and the skin of the muzzle may become cracked and very sore.

On auscultation in the early stages a slightly roughened sound may be heard at the base of the trachea and in the bronchial area of the thorax. These sounds become more pronounced as the inflammatory exudate increases, and are called *moist râles.* These generally appear about the third day of the disease and may remain present throughout. In severe cases there may be an absence of sound in areas beyond the bronchial zone due to medium or smaller bronchi becoming completely occluded, thus preventing the access of air into the air vesicles. In other zones there may be a compensatory emphysema which is indicated by increased vesicular sounds. As the condition improves, sounds may again be heard in the areas where there was loss of sound. This is due to the breaking down of exudate, and the term *secondary râles* is applied to them. At this time bronchial discharge may rapidly become more copious, and when very excessive the term *bronchorrhœa* is applied to the condition.

On percussion dulness of sound will be revealed in the functionless areas, and increased resonance in the emphysematous areas.

Prognosis.—Acute bronchitis may terminate in resolution or death, or it may result in chronic bronchial catarrh and lead to permanent thickening of the mucous membrane of the bronchial tubes with increased susceptibility to catarrh. It may also result in asthma or in pulmonary emphysema, while many horses become permanent roarers. In animals in the prime of life uncomplicated bronchitis is not a very serious condition, but in very young or very old animals it is much more unfavourable, and may pass on to pneumonia or pleurisy and terminate fatally.

Treatment of Acute Bronchitis.—Much depends upon the case being treated early. Unfortunately, in many cases animals are allowed to become seriously ill before treatment is adopted, and in the case of horses they may be worked for several days, thus considerably reducing the prospects of a good recovery. The best possible hygienic conditions should be provided in all cases of respiratory diseases including bronchitis. The patient should thus be placed in a comparatively warm place with a *plentiful supply of fresh air.* Clothing and bandages should be applied to the larger animals, while in short-coated dogs a coat should be put on or the body wrapped in a woollen bandage. Medicated inhalations such as are recommended for nasal catarrh and laryngitis are particularly useful in bronchitis, as they stimulate the bronchial mucous membrane and facilitate the removal of the inflammatory exudates. The application of hot cloths to the chest wall with or without turpentine sprinkled over them afford relief, or alternatively the application to the chest wall and base of the neck of a spirituous stimulating liniment which will not leave the chest wall wet, such as lin. camphoræ ammon. 1 part to lin. saponis 12 parts. The cautious use of a mustard plaster may also be found serviceable, but in this case the hair must first be removed, consequently it is not so useful as the former applications.

Medicinally, stimulating expectorants and internal antiseptics are indicated, while in cases where the cough is very distressing antispasmodics will be found very useful. **In the horse** the administration of drenches should be discountenanced. The agents may be administered either in the form of electuary or in bolus or in the drinking water. As electuary powdered squills, camphor, and extract of belladonna will be found useful, while in bolus may be used quinine sulph. ʒi., ammon. carb. ʒii.-iii., and powdered nux vomica ʒi., which may be administered twice daily. In the drinking water may be used potass. iodide ʒii. or sulph. of magnesia ℥ii.-iv., with potass. nitrate ʒii.-iv. **In cattle,** however, it is usually necessary to drench, and the same agents may be used as are recommended for horses.

In the smaller animals drenching is also not so objectionable, although the administration in the form of pill or powder will often be found more convenient. It is also frequently possible by careful selection of medicines to get dogs to take the remedies voluntarily in milk or drinking water, and it goes without saying that this is the best method when it can be carried out. A selection may be made from the following, namely: Syrup or tincture of squills; glycoheroin; small doses of chlorodyne; salicylate of soda; aceto-salicylic acid; potassium iodide; quinine sulphate; pulv. ipecac. co. (Dover's powder).

The administration of remedies intratracheally is often also recommended, and if done with discretion there is no doubt much to be said for this method. Such agents as Dieckerhoff's solution of iodine (I 1, KI 5, H_2O 100), turpentine with a little chloroform, creosote, and formalin are recommended, and the two latter have been specially recommended in cases where the bronchial discharge is putrid in character. The hypodermic injection of nuclein is of great service in many cases of catarrh of the respiratory tract.

The diet in bronchitis must be somewhat laxative and tempting, dusty foods should be avoided, and a good supply of water should be provided.

Chronic Bronchitis.—Chronic bronchial catarrh is most frequently a sequel to acute bronchitis, but in some cases it is slow and insidious from the outset, especially in the case of old animals suffering from general debility.

Symptoms.—The common symptom is an occasional cough, particularly brought on by changes of temperature or a little exertion. The cough is usually somewhat moist, and there is a nasal discharge which is very tenacious, and may be expelled intermittently in flocculent masses. In the horse, when there is a chronic thickening of the bronchial mucous membrane, respiration becomes audible on light exercise, and horses so affected are termed "thick-winded" or "wheezers."

Bronchiectasis may also be met with in chronic bronchitis. This consists of a dilatation in the course of a bronchial tube, and constitutes a cavity in which a muco-purulent discharge may collect. On auscultation the bronchial sounds are abnormally loud, and in the case of bronchiectasis air may be heard passing through the exudate in the dilated area. The frequent coughing often causes some degree of emphysema, which may be detected on auscultation, and also by increased resonance on percussion. In chronic bronchial catarrh there is rarely any alteration of temperature or pulse, but there is usually great loss of condition; and anæmia may be set up. In all species in chronic bronchitis the pulmonary blood pressure is raised, with a tendency to the production of dilatation and hypertrophy of the right side of the heart with its concomitant symptoms.

In cattle, chronic bronchitis is often difficult to differentiate from pulmonary tuberculosis except with the aid of the tuberculin tests.

Treatment.—Recovery from chronic bronchitis is difficult to obtain, but the condition may be considerably relieved. Medicated inhalations should be persisted in, and internally the best results are generally obtained by the administration of a course of iodides such as potassium iodide, Donovan's solution (iodide of arsenic and mercury 1 per cent solution), and iodide of iron. The addition of squills and nux vomica will also be found very useful. In the case of the smaller animals "cod-liver oil and malt" emulsion is very useful in chronic bronchitis.

Bronchial Asthma

By the term asthma is meant a paroxysmal dyspnœa which frequently arises quite suddenly, and after a variable period may pass away again equally suddenly.

Etiology.—In the majority of cases asthma appears to be due to a highly sensitive or irritable condition of the bronchial mucous membrane which in many cases may be a sequel to a previous attack of bronchitis. In asthma there is a sudden spasm of the bronchial muscle causing a narrowing of the bronchial tubes, bringing about respiratory distress. In some cases it is highly probable that the bronchial spasm may be set up reflexly through the vagus nerve as a result of indigestion (dyspeptic asthma). Peripheral irritation may also set up attacks of asthma, such as irritants on the skin and sudden changes of temperature, as the changing from a warm to a cold atmosphere and *vice versa*. The writer has seen acute attacks of asthma in two cases in horses, following excision of the laryngeal ventricle for roaring. In each of these cases there was violent spasm of the tracheal muscle, which causes a very great narrowing of the lumen of the trachea to about one-fourth its normal calibre. In one instance a temporary tracheotomy tube was inserted with difficulty, while in the other case the symptoms passed away in about ten minutes without necessitating tracheotomy. Many cases of paroxysmal dyspnœa diagnosed as asthma are due to defective heart, such as incompetence of the mitral valve and dilatation of the right ventricle. Such cases are sometimes designated **cardiac asthma.** Again spasmodic dyspnœa may occur in animals affected with pulmonary emphysema, as in broken-wind in horses. It may be that in some of these cases the emphysema is the primary cause of the spasmodic dyspnœa, or that the dyspnœa itself due to spasm of the bronchial muscle may be the exciting cause of the emphysema. In the human subject heredity is regarded as an important factor, but there is very little evidence of hereditary transmission of asthma in the lower animals, except perhaps occasionally in the case of toy dogs.

Anatomical Changes.—Frequently on examining the bronchi of asthmatic subjects no bronchial lesions can be found, while in some cases a chronic bronchitis may be discovered. There are frequently, however, secondary changes met with, such as emphysema in some parts of the lung and some dilatation of the right side of the heart.

Symptoms.—The attacks as a rule come on quite suddenly in the form of intense dyspnœa. They may occur when the patient is at work or at rest. The patient stands with elbows abducted and fore-legs wide apart. The head and neck are outstretched, the nostrils widely dilated, and in many cases in dogs the hind quarters are somewhat depressed with hocks semi-flexed. The dyspnœa is both inspiratory and expiratory, and the chest wall and abdominal muscles are both brought into excessive play. In the majority of cases bronchial sounds are usually audible without auscultation. In the horse there may be a slight cough, which becomes more pronounced in bad cases. A slight nasal discharge may also be present, but is never very copious unless chronic bronchitis is co-existent. In the smaller animals, particularly the dog, the suddenly occurring symptoms are often very distressing to watch. There is a very characteristic cough, a prolonged dry cough, which, to the writer, somewhat fancifully perhaps, often more or less resembles the sound produced by the sawing of wood. In all species the temperature is usually normal throughout, the pulse is frequent and hard, the mucous membranes are somewhat dusky and bluish, particularly in the height of an attack, owing to interference with oxygenation of the blood. There is usually some irregularity of the bowels in asthmatical subjects with a tendency to constipation. During the course of an attack the patient has usually a rather anxious, drawn expression of the countenance. After the symptoms have been in existence a varying time, which may be minutes or hours, or even several days, they may suddenly cease, and the patient again appears to be perfectly normal. Recurrence may occur at varying intervals. It may be a few minutes, or hours, or even days, and in some cases there may be no recurrence for weeks.

Treatment.—The attacks may be cut short in many cases by inhalation of amyl nitrite or burning nitre paper—brown paper previously soaked in a saturated solution of potassium nitrate and dried. The patient should be placed in an airy place, and an equable temperature should be maintained where possible for susceptible patients. Internally, at the time of an attack, sulphuric ether may be administered hypodermically, and in a large number of cases acts almost as a specific. In other instances sedatives are necessary, such as bromide of ammonium or potassium, chloral hydrate, morphia or belladonna. In susceptible subjects attacks can be warded off by the administration of a laxative, and attention to the diet, which should be of a non-irritant, easily digestible character. Calomel is one of the best agents for this purpose, and may be given in small repeated doses to horses or dogs three or four times a week. In the horse the addition of a small amount of linseed oil ℥ii.-iii. to the morning feed will often be found useful. A course of arsenic and antimony tartrate and digitalis, together with nux vomica, will frequently prevent a recurrence of attacks. In the dog, syrup of chloral and ammonium bromide in combination will afford relief in bad cases, while in less acute but persistent cases excellent results are obtained by the combination of sulphuric ether, tincture of digitalis, tincture of nux vomica and aromatic spirits of ammonia. At other times ipecacuanha, glyco-heroin, chlorodyne, syrup of squills and compound tincture of camphor will also be found useful either individually or in combination.

Dosage.—See pp. 544-6.

Diseases of the Lungs

Pulmonary Emphysema; "Broken-Wind"

This is a condition in which there has been over-distension of the air vesicles in the lungs, which may or may not have resulted in rupture of the walls of the vesicle. The condition is usually divided into—

(1) Vesicular emphysema.
(2) Interstitial or interlobular emphysema.

Vesicular Emphysema.—This is often described as being acute or chronic.

Acute vesicular emphysema is a condition in which the lungs have been over-inflated owing to violent inspiratory efforts. The walls of the vesicles have been over-stretched, but not actually ruptured, and the normal power of elastic recoil of the lungs is impaired.

Chronic vesicular emphysema is a much more serious condition, in which the alveoli have been permanently over-distended and the walls in many instances ruptured. This is the condition that is usually described in the horse as "broken-wind."

Interstitial emphysema is a condition in which air is present in the interlobular and subpleural connective tissue. The air passes into these situations through some rupture of the air vesicles, and may extend over a considerable area.

Causes of Vesicular Emphysema.—Vesicular emphysema may be caused either by forced inspiratory efforts or by repeated violent expiratory efforts, and especially in those cases where there is already some weakness of lung tissue. *Violent expiratory effort* occurs in cases of chronic cough and chronic bronchitis. The chronic cough may be due to asthma or in many instances to reflex action through the pneumogastric nerve, and in this way it may be brought about by indigestion. In horses it is commonly asserted to be due to feeding excessively on sainfoin, lucerne, old vetches, and dusty hay. Heavy feeding before exercise is also said to cause broken-wind, especially in horses in soft condition. *Forced inspiratory effort* occurs in asthma and may bring about emphysema. It may also occur as the result of pneumonia or of pleurisy with effusion. In each of the latter cases considerable areas of the lung are functionless owing either to consolidation or to collapse, and as a consequence other areas of the lung may become over-distended and their vesicles ruptured. This is sometimes called **compensatory** or **vicarious emphysema.**

Anatomical Changes.—In acute vesicular emphysema the chief feature is observed immediately the chest is opened. The lungs appear to be unusually voluminous or increased in bulk owing to the fact that they do not collapse to the proper degree. They are usually also paler in colour than normal. In chronic vesicular emphysema a varying degree of lung is affected, but in the majority of cases it is limited to smaller areas than acute vesicular emphysema. The common situations are the apices of the lungs and the lower and posterior edges. The affected patches are paler in colour than normal, are soft and elastic, but appear to be abnormally bulky. The condition being due to rupture of air vesicles resulting in the fusion of several air sacs, shows itself in the form of small adjacent swellings which may attain the size of a pea. In the horse, with so-called broken-wind, emphysema is more extensive and may be met with to greater or less degree all over the surface of the lung. This condition in the horse is nearly always associated with some degree of dilatation or hypertrophy of the right ventricle, owing to the increased blood pressure resulting from the destruction of lung tissue. In interstitial emphysema, the air having escaped into interlobular spaces causes slight vesicular swellings on the surface of the lung, which may be as large as a pea, and which can be moved along by pressing them with the finger. This condition is most frequently met with in the ox.

Symptoms of Emphysema.—In the early stages it is often difficult to detect any abnormal changes in emphysema of the lungs, but as the condition becomes more extensive symptoms arise mainly in the nature of dyspnœa or difficult respiration which becomes more exaggerated on exertion. In early cases there is an occasional harsh dry cough which becomes more pronounced on exercise, and later becomes more or less characteristic. The so-called broken-winded cough is weak, short and shallow, and is occasionally accompanied by a very slight trickle of nasal discharge. The dyspnœa of broken-wind is mainly expiratory, owing to the loss of the elastic recoil of the lungs which necessitates exaggerated abdominal contraction during expiration. The effect of this is to produce a double expiratory flank movement which becomes very marked on exercise, and continued exertion soon brings about respiratory distress. On percussion of the chest wall increased resonance is noted, particularly over the affected areas, such as the inferior and posterior margins of the lungs, while in bad cases of broken-wind the increased resonance may be observed over the entire chest wall. On auscultation the inspiratory murmur is softer than normal and may be associated with some crepitus, which is most marked in interstitial emphysema, while the expiratory murmur is prolonged owing to the diminution in the elasticity of the lung. Horses affected with broken-wind are usually unthrifty in appearance, have a pendulous abdomen and hollow flanks, while indigestion and tympany with the passage of flatus are frequently met with. The latter often occurs during coughing, but is not infrequent at any other period. The temperature in emphysema is usually normal and the mucous membranes are dusky or cyanotic. As a result of the increased blood pressure in the lungs dilatation of the right side of the heart may be set up with duplication of the second cardiac sound, and also resulting in some œdema of the dependent parts and of the extremities. Animals so affected are incapable of any prolonged exertion, and in the case of horses may become quite unworkable and are occasionally subjects of palpitation.

Prognosis of Broken-Wind.—Recovery from broken-wind is impossible. The condition in almost all cases is gradually progressive. Under favourable conditions, however, affected animals may remain workable for years if undue exertion is avoided. Animals so affected appear to be very susceptible to bronchial catarrh, and the presence of such affections considerably prejudices the patient.

Treatment.—*Prevention* in the case of horses is of the utmost importance, particular care being directed towards the feeding and general management. Horses should be fed frequently, and should never be given a big meal immediately before exercise. Moreover, forced exercise should never be given to horses in soft condition. Suddenly occurring broken-wind has not infrequently followed the neglect of this precaution in the hunting-field in the early part of the season. Such food-stuffs as sainfoin, lucerne, vetches, and clover should be used with discretion. It is rarely advisable to use any of these in higher proportion than one part to two or three parts of hay. Otherwise they set up irritation of the alimentary tract and reflexly produce a chronic cough which may develop into broken-wind. Any catarrh of the respiratory tract, however slight it may appear, must be regarded seriously and as a potential cause of broken-wind. Animals so affected should therefore not be worked until recovery is complete.

Curative measures are out of the question, but *palliative treatment* may be adopted with a good measure of success. Animals that are not too far advanced may be rendered serviceable for a considerable time. Especial attention should be paid to the feeding and to the condition of the alimentary tract. All long food should be avoided and care taken that the animal does not eat his bedding. If he is inclined to do so a muzzle must be used. Hay should therefore be given chopped and mixed with the corn and damped. To it may be added with advantage linseed or cod-liver oil, ʒii.-iii. daily in the morning feed, while an occasional linseed mash once or twice a week will be found beneficial. Various medicinal agents have been used with varying success. One of the best combinations consists of white arsenic gr. v., antimony tartrate ʒi., and sulphate of iron ʒii. This should be given daily for five or six days, followed by an interval of two or three days and repeated. As the condition is chronic, so the medicinal agents must be persisted in. Hoare has found the following combination useful:

Powdered extract of aconite	gr. v.
Antimony tartrate	ʒii.
White arsenic	gr. x.
Strychnine sulphate	gr. ss.
Veratrine	gr. i.ss.

This he recommends to be given in the food every evening. Vergotinin, which is a combination of veratrine, ergotinin, and strychnine with glycerine, is efficacious in many cases given once daily in the food. Professor Dick used to claim excellent results with the daily administration of opium, calomel, and digitalis. These various agents undoubtedly tend to relieve the symptoms of broken-wind by their action on the heart and circulation, on the bronchial mucous membrane and on the alimentary tract, but attention to feeding and careful general management are of paramount importance.

Fraudulent measures are often adopted by dealers and others to reduce the chief symptoms of broken-wind in horses offered for sale. One of such methods consists in the administration of lead shot, ½ lb. to 2 lbs., according to the size of the horse, mixed with lard and administered in bolus, the animal at the same time being kept on short rations. Other agents employed include aconite, belladonna, stramonium, tincture of opium, and tartar emetic. These agents all more or less reduce the double expiratory flank movement, and the effect may last for a day, but is followed by more intense dyspnœa owing to respiratory depression when the effects have passed away. When these measures have been adopted it is often very difficult to detect broken-wind in horses, and any respiratory distress, even without the double expiratory flank movement exhibited on exertion, should be regarded with grave suspicion. In such cases Hoare suggests that the horse "should be kept under observation until he has cooled down and should then be allowed cold water and a feed of hay. If affected with broken-wind the diagnostic symptoms will now appear in spite of the deceptions practised."

It is also stated that broken-winded mares advanced in pregnancy often show no double expiratory flank movement on exercise. Suspicion in such cases is only aroused by the character of the cough which may be present and may be supported on percussion and auscultation of the heart and lungs.

Congestion of the Lungs; Pulmonary Hyperæmia; Pulmonary Engorgement

This is a condition in which there is an abnormally large amount of blood in the lungs. It may be brought about by anything which increases the flow of blood to the lungs without a corresponding outflow (active hyperæmia), or anything which diminishes the outflow without a corresponding reduction of the inflow of blood (passive hyperæmia).

Causes or Etiology.—*Active congestion* is frequently the first stage in the development of pneumonia, and is therefore brought about by similar causes, such as:

(1) Bacteria gaining access either by inhaled air or carried to the lung in the blood-stream.

(2) Chemical irritants, such as medicines or food substances or saliva, passing down the trachea and aspirated into the lungs.

(3) Exposure to cold and inclement conditions may be concerned in the causation of congestion of the lungs, as in the case of bronchitis (see p. 381).

Passive congestion is frequently due to cardiac debility and consequent loss of driving power when the animal is put to great exertion. In this way blood may be driven into the lungs by the right heart with insufficient force to drive it through to the left heart. The latter is in turn unable to rise to the demand placed upon it to drive the blood through the greater circulation, and becoming over-distended, still further aggravates the condition of congestion of the lung. This condition frequently follows sharp exercise in horses not in good condition, and is seen in its most acute form in the hunting-field in the early part of a season after a long run. Passive congestion is also met with in mitral insufficiency and in dilatation and fatty degeneration of the heart. It may also be produced by pressure on the pulmonary veins, as in pericarditis.

Hypostatic congestion of the lungs may also occur. This is due to animals remaining in a recumbent position for a considerable time; it is a gravitation congestion, and involves principally the apex and the lower portion of the lungs.

Anatomical Changes.—The vessels of the lung are engorged, and the affected part or the whole lung is found to be more voluminous than normal. The lung does not collapse in the ordinary way. It is found to be very dark red and in many instances almost black. When incised the cut surface is moister than normal, and a quantity of blood and blood-stained froth escapes from the cut surface. The bronchi also frequently contain froth, which is sometimes blood-stained. A portion of the lung cut out and placed in water will still float, thus differentiating it from consolidation of pneumonia. The lung tissue in acute congestion very rapidly decomposes and breaks down. The heart is also distended with dark-coloured blood, which is usually imperfectly coagulated.

Symptoms.—In acute congestion the symptoms are very alarming and are associated with great distress. If it occurs in the hunting-field the horse is practically "run to a stand-still," and if he is pressed is likely to fall. If pulled up, there is noticed very exaggerated blowing respiration, heaving of the flanks and chest wall, legs outstretched and elbows abducted, head and neck extended, nostrils dilated. There is frequently staggering and a tendency to fainting, and the horse may even fall at this point and rise again. The patient usually breaks out into a cold sweat, the body surface and extremities are very cold, and the mucous membranes a dull slaty bluish colour. The pulse is hard, small, weak, and very indistinct, almost imperceptible. There is frequently a jugular pulse, the right side of the heart being over-distended. The temperature is slightly elevated but rises rapidly, and may soon become elevated to 106° F. or 108° F. In cases of acute congestion there is a watery nasal discharge which may be blood-stained. There is also an occasional short dull cough. In acute cases the horse may stand in one position with a marked indisposition to move, and in this respect somewhat resembles an animal with laminitis. On auscultation the condition varies according to the severity of the attack. In some instances there is an increased vesicular murmur, but in the more serious attacks respiratory sounds are considerably reduced and sometimes quite inaudible. The heart is tumultuous and arrhythmical. On percussion there is reduced resonance over the entire lung. In favourable cases recovery may take place in twelve hours, and the congestion may pass off almost as quickly as it arose. The heart improves in tone, the jugular pulse abates, the arterial pulse becomes more distinct and the respirations more easy. In other cases which at first appear to be no more severe, death will occur suddenly in from twelve to twenty-four hours from asphyxia and cardiac failure. In other cases the patient may partially recover, and ultimately succumb in several days from œdema of the lungs. In other cases still, pneumonia may supervene.

Prognosis.—Prognosis in acute congestion of the lungs should always be withheld. It is quite impossible in the acute stages to foretell either course or termination of the attack, while cases which appear likely to recover develop into pneumonia and are ultimately fatal. This is especially likely to occur in those cases which are regarded too lightly and where partial recovery is followed by relapse. In other cases œdema of the lungs may arise and prove fatal, while in a number of cases in horses, laminitis may be set up owing to congestion of the sensitive laminæ in the early stages of the attack. In this respect the hind feet are said to be most frequently affected.

Treatment.—The patient must immediately be placed where he can obtain a plentiful supply of fresh air. If the congestion occurs in the hunting-field the head should be turned to the wind, and all obstructions to free and easy respiration, such as girths and throat lashes, should be released or removed. Immediate relief in acute congestion is best procured by venesection, or bleeding from the jugular vein, which rapidly relieves both the heart and the lungs. The superficial circulation should be stimulated by friction of the limbs and the application of rugs, hood, and bandages. Diffusible stimulants should next be administered, and probably the best of these is sulphuric ether ℥ss. in the case of a horse, or m. x. for the dog, which should be injected hypodermically. The inhalation of amyl nitrite also relieves the heart and internal congestion by increasing superficial circulation. Aconite may also be found serviceable, but must be used with caution on account of its depressant after-effect on the heart. It should be only administered when the heart is in fairly good tone. Other useful stimulants include digitalin, camphor, caffeine, and strychnine, all of which may be administered hypodermically. The application of stimulants to the chest wall, such as a weak mustard plaster, or better still the application of hot cloths, affords considerable relief. It is inadvisable to forcibly administer any remedies by drenches in congestion of the lungs or other respiratory affections if it can possibly be avoided, but it may happen that professional assistance is not at hand for hypodermic administration of suitable remedies. In such cases, such as those occurring in the hunting - field, the *careful* administration of whisky or brandy ℥viii.-x. well diluted may be instrumental in saving the patient. The horse should be got to the stable by means of an ambulance if possible when the distress has somewhat abated. In cases where the acute symptoms pass away fairly rapidly, it must be remembered that the animal has been entirely thrown out of hard condition, and a long course of convalescence is necessary, followed by a full conditioning course. There is a great danger of recurrence if exercise is permitted too soon, and it is then frequently followed with pneumonia with fatal termination. During convalescence a course of cardiac tonics and stimulating expectorants should be given, such as nux vomica, digitalis, squills, and ammonia. Diet must be concentrated, nutritious, and slightly laxative, and in all cases dusty foods and dusty bedding should be avoided.

Dosage.—See pp. 544-6.

Œdema of the Lungs; Pulmonary Œdema

This is a condition in which there is extravasation of serous fluid into the lung tissue and air sacs. It is generally brought about by congestion of the lungs and debilitated circulation. It may be due to toxins circulating in the blood, and in this way may occur in nephritis, and in the course of a number of specific diseases, especially septicæmias, such as anthrax, East Coast fever in cattle, Cape horse-sickness, etc. It may also occur in purpura hæmorrhagica, and in anæmia from any cause.

Anatomical Changes.—When the chest is opened the lung does not collapse to its normal extent, and when cut a frothy mucus oozes from the air vesicles and the bronchial tubes, and also occupies the larger bronchi and trachea. When the œdema accompanies acute congestion the froth may be somewhat hæmorrhagic. If the post-mortem examination is delayed, a quantity of frothy mucus may escape from the nostrils, and in some instances this may be very copious, as in Rhodesian fever and in Cape horse-sickness. In each of these cases the froth may become heaped up round about the nostrils almost like a small heap of snow.

Symptoms.—Œdema of the lungs usually develops very rapidly, and gives rise to very great dyspnœa and may lead to suffocation. The nostrils are widely dilated, and a foamy discharge may be coughed up and expelled from the nose. On auscultation in slight cases there is increased vesicular murmur, but this soon becomes lost owing to the blocking of the air vesicles with œdematous liquid. In the bronchial area râles may be readily observed. On percussion dulness is revealed. Œdema of the lungs is generally very rapidly progressive, and in the majority of cases terminates fatally in a very short time, the animal practically drowning in his own serum.

Treatment.—Precisely the same medicinal measures may be adopted as are recommended for acute congestion of the lungs, but results are likely to be very disappointing, as the condition is rapidly fatal in a vast majority of cases.

Hæmoptysis ; Bronchorrhagia ; Pulmonary Hæmorrhage

This is a condition of hæmorrhage in the trachea, bronchi, or the lung tissue. The causes are varied, but in the majority of cases it is due to an acute pulmonary congestion. It may thus occur in acute congestion of the lungs and pneumonia resulting from over-exertion, as in hunters and racers. It may also occur during the course of various specific affections, such as anthrax, purpura hæmorrhagica, more rarely tuberculosis, and also from ulceration of the trachea in glanders. Pulmonary hæmorrhage may also be due to traumatic causes, such as broken ribs which wound the lung tissue, bayonet wounds, and gunshot wounds.

Symptoms.—In cases where the hæmorrhage is very limited, the only thing noticeable may be a slight trickle of blood from the nostrils with or without marked coughing. When, however, the hæmorrhage is excessive, dyspnœa is set up with spasmodic coughing and the discharge from the nostrils of more or less copious quantities of blood, which may be churned up into a bright red froth. The condition appears urgent and the patient has an anxious expression, often breaks out into perspiration, and may stagger and fall and die from suffocation.

Treatment.—In mild cases recovery may be expected. The patient should be placed in a quiet, cool, well-ventilated box, and all forms of excitement should be carefully avoided. The internal blood pressure should be reduced by stimulating the external circulation as far as possible, by the application of warm clothing and the administration of salines and nitrites. The administration of the classical hæmostatics is of doubtful value. Agents such as ergotin, hydrastis, and adrenalin are frequently recommended, but as these agents all increase blood pressure they are likely to intensify the hæmorrhage rather than reduce it. Opium or morphia gives better results. They reduce the heart's action, lower blood pressure, quieten the cough, and have a general tendency to make the patient more restful. Following the morphia a smart dose of a saline purgative should be administered, thus further reducing blood pressure. If the hæmorrhage is very severe, sterile normal saline solution should be injected subcutaneously.

Pneumonia ; Inflammation of the Lungs

There are several forms of pneumonia met with in domesticated animals. They are sometimes classified according to the cause of the condition, or according to the distribution and nature of the lesions produced :

Catarrhal Pneumonia.—In this form proliferation and desquamation of epithelium is the most prominent feature. The lesion thus formed is generally associated with a similar condition of the small bronchial tubes, and it is consequently in such cases called **broncho-pneumonia**. It is also called **lobular pneumonia**, since the lesions have a lobular disposition. If large areas of the lung are affected in this form of pneumonia, it is generally due to a number of contiguous small areas being simultaneously affected.

Croupous Pneumonia.—In this form the chief feature is seen in the character of the inflammatory exudate, which is of a fibrinous or croupous nature. This form affects large areas of the lung, and it is consequently also called **lobar pneumonia.**

Interstitial Pneumonia.—In this form there is an increased formation of fibrous tissue in the lung. It is usually a chronic condition. It is also known as **fibroid pneumonia** and **pulmonary cirrhosis.**

In some forms of pneumonia the inflamed tissues become invaded with septic organisms setting up gangrene; this is called **septic** or **gangrenous pneumonia.** Where the pneumonia is associated with pus formation in the lungs it is called **purulent pneumonia.** When inflammation of the lungs is set up by parasites it is called **parasitic pneumonia.** When the condition is due to faulty drenching and the passage of foreign substances into the lungs by way of a trachea the condition set up is called **drenching pneumonia.** This form is frequently septic in character.

Catarrhal or Lobular Pneumonia.—*Etiology.*—Catarrhal pneumonia is brought about by irritants gaining direct access to the alveoli of the lungs. These include chemical irritants, such as medicines and food materials, which have passed down the trachea into the small bronchi, either as the result of faulty drenching or as the result of intratracheal injection of irritant substances insufficiently diluted. The condition may also be due to the inhalation of irritant gases, such as chlorine gas, sulphurous acid, formalin vapour, etc. Animal or vegetable parasites may also set up broncho-pneumonia. The animal parasites most frequently causing broncho-pneumonia are the *Strongylus rufescens* in sheep. Of the vegetable parasites various moulds, such as aspergillus, have been responsible for pneumonia. By far the commonest causes of pneumonia are bacteria, which may either be inhaled or be carried to the lungs by the blood-stream. In this way pneumonia may be secondary to infectious diseases, as in the case of distemper in the dog, tuberculosis, glanders, influenza, etc.

There are various *predisposing causes* of pneumonia, such as advanced age, debility, anæmia, and exposure to cold. Cold is often thought to be an exciting cause of pneumonia, but it probably acts by reducing the natural powers of resistance and thus enabling organisms to penetrate more easily and so set up inflammation of the lungs.

Anatomical Changes.—The principal feature of this condition is the presence in the air sacs of an inflammatory exudate consisting principally of catarrhal cells and some leucocytes. A similar exudate is also present in the small bronchial tubes. The lesions are localized, affecting the lobules of the lung. The extent is variable; in some cases it may affect only a few lobules, or it may be widely disseminated. The affected areas are more or less solid, and do not collapse when the chest is opened. They consequently stand up above the level of the surrounding lung tissue. The affected patch is firmer than normal and contains no air. The colour varies from greyish red to greyish yellow. The surrounding lung tissue is usually affected with compensatory hyperæmia. Other areas may be affected with collapse, and sink below the level of the surrounding tissues. Other parts of the lung may show areas of emphysema. On cutting the inflamed areas a thick yellowish-white exudate can be squeezed from the air sacs and small bronchi of the cut surface. On microscopic examination the alveoli are found to contain an essentially cellular exudate, and only rarely is fibrin present. The pleura over the affected patch is often somewhat roughened, while the bronchial lymphatic glands are swollen and juicy. Where the condition is due to tuberculosis caseation is usually present.

Symptoms.—Catarrhal pneumonia is in the majority of cases preceded by bronchitis. In fact, the symptoms are frequently those of an exaggerated form of bronchitis. The temperature rises 2° or 3°, varying according to the case, but is rarely very high. The respiration is accelerated and difficult, and is usually associated with a moist cough and a catarrhal nasal discharge. The head is outstretched and nostrils dilated, while in advanced cases in the dog the lips and cheeks are drawn in during inspiration and inflated during expiration. There is also frequently some discharge from the eyes, and the mucous membranes have a bluish tinge. The pulse is accelerated and fairly strong. The appetite is suppressed, and in milch animals lactation is diminished, while rumination in cattle and sheep ceases. There is usually some degree of constipation. On auscultation in the earlier stages there are increased vesicular murmurs and bronchial sounds. As the condition advances the vesicular sounds may be completely lost, while the sounds of the larger bronchial tubes become increased. As the condition improves, vesicular murmurs may become re-established owing to the breaking down of exudate. This feature is generally associated with increased coughing and nasal discharge, but the patient is usually somewhat brighter. On percussion, coughing may be set up, and dulness is revealed over the affected areas. In catarrhal pneumonia the patient loses condition very rapidly, and horses so affected rarely lie down, cattle do so occasionally, while dogs usually rest on the sternum.

The course of catarrhal pneumonia is very variable. It may prove fatal in a few days, or it may last for several weeks to be followed by recovery. When the case is taking a favourable turn the respiration becomes easier and the temperature subsides gradually. The cough is easier but still maintained, and the nasal discharge is increased for a time. There is also an increased brightness in the patient, who looks for food and will take a little at a time, while cattle and sheep occasionally chew the cud. If the improvement is maintained, recovery may be complete in ten days or a fortnight. Less favourable cases may be followed by chronic interstitial pneumonia, or by caseation as in tuberculosis, or a relapse may occur. In other cases death follows from suffocation or from cardiac failure or from the absorption of toxic products generated in the inflamed areas. More rarely gangrene of the lungs may be set up.

Treatment.—This must depend to some extent upon the primary cause of the condition ; if it occurs during the course of any specific disease, such as distemper of the dog, appropriate treatment for that affection must be adopted. Every case of catarrhal pneumonia should be regarded as a possible source of infection, and consequently isolation should be practised. The best hygienic conditions possible should be procured, affording a plentiful supply of fresh air and, if possible, an equable temperature. Clothing should be applied in suitable cases. Relief is also afforded by the application of hot cloths to the chest wall or a mild mustard plaster or a mild blister. Medicated inhalations should be resorted to as recommended for bronchitis. The administration of medicinal agents by drenching should be avoided, and such remedies should be selected as will either be taken voluntarily or can be given by other means, either in the form of bolus or pill or electuary, or administered hypodermically. For the horse and cattle a very useful combination consists of quinine sulphate ʒi., potassium iodide and ammonium carbonate āā ʒii., and pulv. nux vomica ʒi. This may be made into a bolus and administered twice daily. Alternatively antifebrin ʒi. with powdered squills and camphor āā ʒii. may be administered in electuary. Salicylates may also be found useful, but owing to their cardiac depressant effect should be combined with a stimulant such as ammonia. Quite good results are also frequently obtained by the administration of magnesium sulphate ℥ii. and potassium nitrate ʒii., which may be given in the drinking water. For dogs the cough may be relieved by the administration of small doses of chlorodyne or by means of compound tincture of camphor, while sodium salicylate, tincture of squills, and aromatic spirits of ammonia will frequently be found a useful combination. In any species where there is any danger of heart failure the hypodermic injection of ether or digitalin, strychnine, or caffeine should be resorted to. The use of nuclein injected subcutaneously will frequently be found of great service in this as in many other catarrhal affections.

Attention to nursing and food is of paramount importance ; the appetite must be tempted, food being offered frequently and in small quantities, and varied as much as possible. For the large animals, boiled linseed, bran mashes, oatmeal gruel, boiled oats, steamed hay, hay tea and milk will all be found useful, while the addition of an occasional sliced carrot, turnip or swede, or a few apples will often stimulate the appetite. When available a little green food should also be offered.

Small animals should be fed frequently and with small quantities. A little finely minced beef or mutton, cooked or uncooked, or horse flesh may be offered. When this fails, a little liver, rabbit, rabbit gravy, or giblets will often tempt the patient to eat a little voluntarily. The water supply should be curtailed, and milk or milk and lime-water offered in its stead. If the patient refuses to feed voluntarily this nourishment must be forcibly administered with patience, great care being exercised so that none of the food passes into the trachea. For this purpose may be used milk or milk with egg, beef essences, or any of the invalid or infant food prepared for the human subject. The combination of cod-liver oil and extract of malt in equal parts, giving a large teaspoonful two or three times a day, is also good.

A long period of convalescence must be allowed. In the case of horses this should extend over several weeks, and the horse should be exercised and gradually brought into work. If this precaution is neglected, relapse is very likely to occur with fatal results.

Croupous or Lobar Pneumonia.—This is the most common form of pneumonia in the horse. It may occur sporadically, or it may occur in the form of a contagious disease, as in epizoötic pleuro-pneumonia of the horse. It is also met with in other species of animals, especially as the result of contagion, as in contagious bovine pleuro-pneumonia and in swine lung-plague. Croupous pneumonia is very rare in the dog, but it is occasionally met with in the cat in the form of an epizoötic.

Considerable doubt has been expressed by various authors as to whether croupous pneumonia in a horse is ever truly sporadic. It is suggested by them that all cases of croupous pneumonia are both bacterial and contagious, and the fact that one may meet with single cases in a stud of horses from time to time is due to a low degree of virulence of the causal organisms. In these cases bacteria may be demonstrated which are indistinguishable from those occurring in epizoötic croupous pneumonia. This view that *all* cases are of the contagious type is one to which the writer is unable to subscribe, although it may be correct in a large number of cases. Sporadic pneumonia of the croupous type undoubtedly is produced in numerous cases as the result of over-exertion when in an unfit condition, as in the case of hunters after a hard run at the beginning of the hunting season. In fact, the condition is set up in similar circumstances as those described under the heading of acute congestion of the lungs of horses. When referring to that condition it was stated that acute congestion due to this cause may pass on in unfavourable cases into pneumonia. When that occurs it is the croupous form of pneumonia that is produced, and the writer has never met with nor does he know of any instance of contagion in a stud following primary cases of this character. Overwork and undue exposure to cold and inclement conditions are predisposing causes to pneumonia of this type, by permitting the invasion into the tissues of organisms frequently found in the respiratory tract but ordinarily non-pathogenic.

Croupous pneumonia may also be produced as the result of inhalation of irritant gases, such as chlorine vapour, etc., and similarly it may be due to the passage of irritant medicinal agents into the trachea on drenching, or by their too concentrated use for intratracheal injections.

Anatomical Changes.—The principal feature in this condition is the presence of a fibrinous or croupous exudate into the air vesicles of large areas of one or both lungs. It is therefore *lobar* in distribution. Pathologically in a typical case croupous pneumonia is described as passing through a series of three stages.

1. *The Stage of Congestion or Engorgement.*—This condition is of very short duration, rarely exceeding one or two days, and consists of acute hyperæmia, in which the lung is found to be dark red in colour and does not collapse to the normal degree. If cut, a quantity of blood and fine froth oozes from the cut surface. At this stage the lung tissue will still float on water. This condition is rarely seen, owing to the fact that it is never fatal at this stage.

2. *The Stage of Red Hepatization.*—This is due to the coagulation of fibrinous exudate in the lungs. The affected lobe does not collapse on opening the chest. When cut the surface is red or greyish red, and has a somewhat granular appearance. If a portion is cut out and thrown into water it sinks, owing to it having become solid. This stage lasts about two days and passes on into the third stage.

3. *The Stage of Grey Hepatization.*—In this stage the lung tissue has become paler than in the second stage, and has become greyish red or greyish yellow in colour. On cutting, the affected part is found to be less firm and more flabby than in the second stage, and it is also less granular in appearance. The affected part will still sink in water.

In the horse it is very difficult to follow this train of changes in the majority of cases of croupous pneumonia. In fatal cases in horses, on opening the chest it is frequently seen that the condition is complicated with pleurisy with effusion. One or both sides of the chest may be affected. The commonest seat is in the lower half of the main lobe of the lung and the apex of the lung. The affected area is extensively hepatized, but the consolidated part is rarely uniform in appearance throughout. It often presents a very granular appearance, and sometimes contains numerous white spots surrounded by dark red areas. The air sacs are filled with leucocytes and croupous exudate or sometimes blood. In many cases there will also be found areas of necrosis which have a grey colour and a fœtid odour.

In croupous pneumonia in cattle and in swine it is common to find the connective tissue of the interlobular septa also distended with a firm croupous or gelatinous exudate producing a condition more or less resembling the veins of marble.

Symptoms.—In the majority of cases, and particularly in the horse, croupous pneumonia is ushered in with a high temperature of 105°-107° F., and this may be the only symptom discoverable for twelve to twenty-four hours. This fact is probably responsible for the high rate of mortality in horses, since the owner or attendant frequently has not realized that the animal is ill, and it has been taken out to work as usual, causing great aggravation of the condition. When this has occurred the case usually terminates fatally. In the early stages the appetite is frequently not interfered with, at any rate during the first day of the attack. The pulse at this time during the early congestive stage is accelerated and much smaller than normal, but as the condition advances the character of the pulse changes, and it becomes quick, frequent, full, and bounding. In unfavourable cases the pulse again undergoes a change, becoming small and weak and lacking in tone, while in favourable cases it diminishes in frequency and improves in general tone. In the earlier stages there are frequently rigors or shivering fits with a cold body-surface and extremities, but this soon gives way to flushing of the superficial vessels and improvement in the external temperature. The internal temperature usually remains high up to the critical stage from five to seven days after the onset of symptoms. In favourable cases the temperature then declines gradually. A sudden fall of temperature is a very unfavourable sign, and frequently occurs twelve to twenty-four hours before death in fatal cases. About the second day of the condition the change in the character of respiration may be noted. The breathing is quickened and blowing, the neck outstretched and nostrils dilated. In the earlier stages the respiration is both thoracic and abdominal, but if the condition becomes complicated with pleurisy the chest becomes fixed and a flopping abdominal respiration is set up. The flank and abdomen become tucked up and the patient rapidly loses condition. The mucous membranes are injected and in some cases have a slightly yellowish tinge. Coughing may be set up, particularly if the patient is excited or made to undergo any exercise, but otherwise coughing is not a very pronounced symptom, and is never so exaggerated as in bronchitis. In most cases there is set up a nasal discharge, which is only very slight and watery in the earlier stages, but becomes more copious, thicker, and viscid in character. In the more advanced stage the discharge becomes yellowish-brown in colour. This is most pronounced when the exudate is beginning to break up and is removed by coughing. If it remains free from putrid odour it is not a bad sign, but if it becomes offensive it is usually an indication of the onset of fatal gangrene. During the course of acute pneumonia a horse rarely lies down, but if he does, it is usually on the affected side unless the case is complicated with pleurisy; in that case if he lies down at all it is on the healthy side during the painful stage, but on the affected side when the pain has subsided. Cattle with pneumonia grunt and grind their teeth. They more frequently lie down than horses, and when they do so it is on the affected side. The bowels are usually somewhat constipated, and the fæces are firm and mucus-coated.

On *percussion* there is loss of resonance over the consolidated areas, particularly in the lower half of the chest. This dull area may extend as the inflammatory changes invade a wider area of the lung. In the non-affected areas there is increased resonance on percussion. On *auscultation* in the earlier stages there is an increased or roughened sound in the vesicular areas during inspiration. This is called primary crepitation. As the air vesicles become filled with exudate the respiratory sounds are lost in the affected part, but there may be increased respiratory sounds in the bronchial tubes leading to the part. When recovery begins to take place the exudate is broken down and vesicular sounds are again produced (median crepitations). At the same time bronchial sounds are increased, and there is an increase in coughing and nasal discharge, but associated with increasing brightness. In consolidation of the lung no respiratory sounds are heard in the part, but there is frequently observed an intensification of the cardiac sounds.

In unfavourable cases when the condition is passing into gangrene there is a sudden fall of temperature and the patient appears brighter and easier. This, however, is soon followed by a fœtid odour of the breath and nasal discharge, and usually terminates fatally in twenty-four to thirty-six hours. In other unfavourable cases the patient becomes weaker, the head is depressed, the mucous membranes become slaty blue, the pulse small, weak, and irregular. In these cases death is likely to follow from heart failure. In favourable cases the crisis at the fifth to seventh day is followed by a gradual fall in temperature and marked improvement in the appetite, the coughing is a little more pronounced and the nasal discharge somewhat increased, but it appears to be much more free and easy. The pulse improves in tone and is less frequent, while the respiration becomes more easy and the flank is seen gradually to drop, the miserable tucked-up appearance of the acute stage gradually passing away.

The probabilities of recovery are in direct proportion to the rapidity with which the condition is recognized, and absolute rest and careful nursing undertaken. In this way one constantly meets with more favourable results in studs managed by observant attendants than in those cases where negligent or less observant attendants have failed to notice indisposition on the part of the patient until it has been ill several days, and in the case of a horse that has been worked during that time.

Treatment.—The patient should be placed at once in the best hygienic conditions procurable, a plentiful supply of fresh air being of paramount importance. In the case of horses and cattle they may be put into a box or shed with one side completely open and protected from rain. Clothing and bandages should be applied to compensate for the lack of warmth in these conditions. It is always preferable in these respiratory cases to sacrifice temperature for fresh air than *vice versa*. Since it is impossible to say definitely whether a case of croupous pneumonia is likely to be of the contagious variety, strict isolation should be practised as a precaution, and the person attending the patient should have nothing to do with healthy animals for fear of conveying infection.

Attention to feeding is the next point in order of importance. As the appetite is not usually totally lost the patient will generally feed voluntarily if it is offered suitable food frequently and in small quantities. It should be of an easily digested character and should follow similar lines as have been recommended in the feeding of patients affected with catarrhal pneumonia. Relief is often afforded by the application of hot cloths or mild counter-irritants to the chest wall, and medicated inhalations may assist the removal of the exudates from the respiratory tract.

Medicinally, the administration of draughts should be avoided, owing to the great danger of aspiration of the agents into the trachea and so aggravating the condition and possibly setting up sepsis. The writer is of strong opinion that far more animals are killed by neglect of this precaution than would have died from the primary affection if they had been left untreated with medicinal agents. Medicines may, and undoubtedly do, in many cases very materially assist recovery by stimulating and maintaining vitality and assisting in the elimination of toxic products. Their use should be directed to these ends. If the case is seen early the administration of a diffusible stimulant associated with a febrifuge will frequently cut short the attack or reduce its intensity. With this end in view, probably the best procedure is to administer hypodermically sulphuric ether and strychnine, and at the same time to give by the mouth antifebrin or a salicylate either in bolus or electuary, or mixed with a small feed. On subsequent days if the patient continues to feed he should be given a combination of powdered digitalis or squills, together with nux vomica and carbonate of ammonia. Horses and cattle will usually take these medicines voluntarily in a small feed. If they will not do so the medicaments may be administered in bolus. The combination of quinine, potassium iodide, nux vomica, and ammonia in bolus as recommended for catarrhal pneumonia will also be found very useful in a considerable number of cases. In many cases the simpler administration of sulphate of magnesia and potassium nitrate in the drinking water gives excellent results, both acting as febrifuge and eliminative. The use of nuclein hypodermically or by the mouth will be found of considerable service in this affection by stimulating vitality of the leucocytes. Collargol is also recommended, and excellent results have been claimed for its use, hypodermically or intravenously. In cases where there is a danger of cardiac failure the use of digitalis is especially indicated, and caffeine and camphorated oil hypodermically will frequently be followed by general improvement. Where the breath is very offensive and there is a danger of the onset of gangrene, carbolic acid or creosote (ʒii.-iii. of 1 per cent solution) or the same amount of Lugol's solution of iodine may be administered intratracheally. Injections should be made very slowly to avoid producing spasmodic coughing, and may be repeated daily for several days. In those cases where pleurisy is developed as a complication fluid should be drawn off from the chest as early as possible. Unfortunately this procedure is frequently delayed until the case is too far advanced for it to be of much service, and consequently many practitioners speak disparagingly of the procedure. The writer is convinced, however, that early puncture is highly beneficial, and he would prefer to resort to it in a mistaken diagnosis of pleurisy than to wait until the condition is well established. There can be no objection to the practice if proper antiseptic precautions are carried out. Moreover, it has been suggested that puncture of the lung in uncomplicated pneumonia will often assist in the absorption of exudates in the inflamed area. The seat of puncture is about a hand's-breadth behind the point of the elbow a little above the spur vein, and the process may be repeated daily if necessary. The use of vaccines and so-called phylacogens has frequently been resorted to, and is strongly recommended by many practitioners, but we have not observed any beneficial effects from their use.

In the rare cases when croupous pneumonia occurs in the small animals the same treatment may be adopted as has been recommended in the treatment of catarrhal pneumonia.

A long period of convalescence must be allowed in horses especially, and one frequently has considerable difficulty in persuading owners of horses that the animal is still unfit for work after apparent recovery and when the patient has been feeding fairly well and improving in general condition for two or three weeks. The period of five or six weeks is the minimum time which must elapse before the horse is put even to light work. Neglect of this precaution is likely to lead to relapse with fatal results, or to set up chronic pneumonia, or even pulmonary emphysema or dilatation of the heart. Complete recovery from an attack of pneumonia in the horse does not appear to predispose to subsequent attacks, as so often appears to be the case in the human subject.

Dosage.—See pp. 544-6.

Chronic Interstitial Pneumonia; Fibroid Pneumonia; Pulmonary Cirrhosis

In this form of pneumonia there is a formation of new fibrous tissue in the lung. It is rarely primary in origin, and is generally a sequel to chronic bronchitis.

Anatomical Changes.—The affected portion of the lung is increased in density and toughness, but it is not usually solid. It, however, contains less air than normal. The colour is variable; it is usually paler than normal and may be greyish white or yellowish grey. The condition as a rule does not affect any large area of the lung uniformly. It is met with principally in the neighbourhood of bronchial tubes and along the course of the blood-vessels. The commonest form of fibroid pneumonia of the lower animals is due to worm infestation, principally *Strongylus rufescens* in sheep, and to echinococcus cysts.

Symptoms.—The condition is a slowly developing one, and does not give rise to very pronounced symptoms by which it can be easily diagnosed. The main symptoms are associated with dyspnœa or breathlessness on exertion, and accompanied by a short cough. It is thus somewhat suggestive of asthma. As in chronic bronchial catarrh in old-standing cases, the patient loses condition, becomes weak and debilitated, and dilatation of the right heart may be set up with its attendant sequels. Fever is not a prominent symptom, in fact in many cases there is no elevation of temperature during the major part of the course, but there may be a periodical and temporary rise of temperature of 2° or 3° F.

On *auscultation* increased bronchial sounds may be observed with dulness of vesicular sounds in the affected pulmonary areas. In widespread cases emphysema of other parts of the lung may be detected. On *percussion* there is dulness of the inflamed areas with increased resonance in the emphysematous parts.

Chronic interstitial pneumonia is sometimes fatal as the result of exhaustion, or the animal may have to be destroyed owing to its being rendered unfit for work as the result of increasing respiratory distress.

Treatment.—Curative treatment of chronic interstitial pneumonia is practically hopeless; palliative measures, however, may be adopted, and on the same lines as those recommended for chronic bronchitis and pulmonary emphysema. By these measures horses can be kept at light work for months or even years. In the case of cattle and sheep with a parasitic invasion of the lungs no medicinal measures are available, and the animals should be fed up for the butcher.

Pleurisy; Pleuritis; Inflammation of the Pleura

Pleurisy is very frequently met with in the domesticated animals.

Etiology.—The vast majority of cases of pleurisy in the lower animals are due to bacteria. Cold is a commonly assigned cause, such as exposure to inclement conditions and a sudden lowering of the external temperature. As in the case of inflammation of other organs and tissues, it is probable that cold is only a predisposing cause weakening the defences of the body against the organisms which constitute the exciting cause of the inflammation. Pleurisy may be *primary* in origin or it may be *secondary* to other conditions. Primary pleurisy is most frequently met with in horses and dogs. Secondary pleurisy may follow inflammation of any of the adjacent organs, such as the lungs, pericardium, or thoracic lymphatic glands, or it may be secondary to peritonitis by extension through the diaphragm. Secondary pleurisy may be due also to invasion from remote parts of the body by way of a blood-stream, as in pyæmia and in septicæmia. Another form of secondary pleurisy is traumatic in origin, as in the case of wounds of the chest wall, fractured ribs, perforation of the œsophagus by means of sharp bones or other foreign bodies in the dog, and also the passage of a foreign body such as a nail or other metal from the reticulum in cattle. In the horse the organism which is most frequently responsible for pleurisy is a streptococcus closely resembling or indistinguishable from the streptococcus of strangles. This may be present without any discoverable lesions elsewhere, but in other cases it may have invaded the pleura from the lungs in broncho-pneumonia, or it may have occurred during an attack of strangles. Another organism frequently causing pleurisy in the horse is an ovoid bacterium which is the cause of contagious pneumonia in the horse. This organism may be responsible for a non-putrid pleurisy. If the condition is accompanied by gangrenous pneumonia the pleurisy is putrid in character owing to the additional invasion of putrefactive organisms. In rare cases the glanders bacillus may be responsible for pleurisy in the horse.

In cattle and dogs and cats the most common cause of pleurisy is the tubercle bacillus; in the first named most frequently giving rise to dry pleurisy, and in the others it is most frequently associated with considerable effusion. In cattle pleurisy is also met with in contagious bovine pleuro-pneumonia and in hæmorrhagic septicæmia, and in dogs in distemper.

Anatomical Changes.—The first change taking place in an inflamed pleura is congestion of the vessels with temporary dryness and dulness (loss of lustre). This is followed by a thickening of the pleura and an exudate into the pleural sac. The quantity and character of the exudate vary very considerably. In the horse the amount may be anything from 1 or 2 gallons up to 10 gallons, and in dogs from ½ pint to 2 or 3 pints, and in cats up to 1 pint. In some cases the exudate is extremely small in amount, and this may occur in chronic pleurisy which has been slow and insidious in onset. In acute pleurisy the invasion is rapid and the exudate is usually copious, but varies as to the amount of fibrin and the number of leucocytes it contains. When the exudate is rich in fibrin it frequently coagulates on the surface of the pleura in the form of a yellowish membrane which can be stripped off. A similar material may be free in the inflammatory liquid. This condition is called a **sero-fibrinous pleurisy,** and is the commonest form met with in the horse. In other cases the liquid contains large numbers of leucocytes, giving it the appearance of pus. This is called a **purulent pleurisy** and the condition is known as empyema of the chest. In some cases the exudate is hæmorrhagic or blood-tinged. The odour of the exudate may be offensive or inoffensive. The former condition occurs in traumatic pleurisy, and also when there is a secondary invasion of putrefactive organisms. In cases of subacute or chronic pleurisy the lung may be found adherent to the chest wall by fibrous adhesions. This is due to the removal of the exudate and the formation of granulation tissue. Extensive adhesions are not very common in the horse, owing to the fact that the majority of cases of pleurisy are fatal. In pleurisy with effusion the fluid causes pressure on the lungs, particularly of the lower border and extending upwards as the volume of liquid increases. This produces pressure collapse of those parts, and may also be responsible for a compensatory emphysema in the parts of the lung not involved.

In the majority of cases pleurisy is unilateral, except in the horse, where, owing to the perforated mediastinum, the majority of cases are bilateral. Unilateral pleurisy in the horse, however, is not infrequent, and the writer has met with purulent pleurisy in which one side of the chest alone was involved and from which 4 to 5 gallons of thick creamy exudate was removed from an incision between the ribs.

Clinically, pleurisy may be regarded as *acute* or *chronic*.

Acute Pleurisy.—*Symptoms.*—The onset of symptoms in acute pleurisy varies somewhat. In the majority of cases there is a considerable amount of pain evinced quite early, the patient appearing very distressed, with an anxious expression and standing still as though any movement caused considerable pain. The patient looks round to the side frequently and may break out into patchy sweats. These symptoms may give rise to mistaken diagnosis of colic in horse, or to laminitis owing to indisposition to move. In some instances it commences as a general indisposition with loss of appetite and dulness and rigors. The mucous membranes are injected and there may be slight catarrhal discharge from the nose and eyes. The temperature is elevated and may be anything from 104° to 107° F., and frequently undulating. The pulse is frequent, small, and hard (wiry) during the primary dry stage ; it later becomes soft and large when the acute stage is passed. In fatal cases the pulse becomes very rapid and weak and almost imperceptible. The respiration is more or less characteristic. It is accelerated and abdominal in character, the chest wall being almost fixed, the flanks and abdominal muscles working excessively (flopping) ; the elbows abducted ; head and neck outstretched and nostrils dilated. While taking the temperature per rectum the anus may frequently be seen pulsating markedly with each respiration. The fixation of the chest wall and the excessive inspiratory movements of the abdominal muscles bring the costal cartilages into prominence, producing what is frequently called the "pleuritic ridge." Pressure on the chest wall or turning round causes pain and flinching, and in horses and cattle grunting, while dogs and cats cry out when picked up. The pain becomes reduced as the exudate increases, but owing to the increased pressure on the lungs the inspiratory effort becomes more exaggerated. The inspiratory effort is far more exaggerated in bilateral pleurisy than when the inflammation is limited to one side of the chest. Pressure of the liquid in the chest frequently produces a jugular pulse together with œdema under the breast and often of the extremities. The presence of a short, sharp, painful cough is observed in the early stages of acute pleurisy, but it is often absent in slowly developing pleurisy ; it is easily brought on by light exertion. Horses and cattle with acute pleurisy rarely lie down, at any rate in the painful stages of the affection ; if they do so it is usually on the healthy side to avoid pressure on the acutely inflamed chest wall, while when the exudation has taken place and the painful condition relieved the patient lies on the affected side in order to reduce the pressure of the exudate on the lung of the opposite side. In this way respiration is less seriously impeded. Dogs and cats usually lie down throughout the attack.

On *percussion* of the chest wall there is a dulness of sound in the lower part of the chest parallel with the height of the effusion, but there is little alteration in the upper part of the chest, unless there is compensatory emphysema, which results in slightly increased resonance. On *auscultation* in the early stages a dry friction sound may be heard over the affected area during both inspiration and expiration. As the lesion extends the friction sounds may be heard along the margin of the lesion. This stage is constantly met with in the smaller animals, but in the horse and cow, owing to the shortness of the period, it is frequently overlooked. After the friction stage has passed there is very little to hear on auscultation until the fluid commences to accumulate in the lower part of the chest causing pressure on the lung and collapse. In this area both respiratory and cardiac sounds are obscured up to the level of the liquid, while movement of the fluid itself may also be heard. In the upper unaffected part, and on the healthy side in unilateral pleurisy, there is increased vesicular murmur. As the condition improves and the exudate becomes absorbed the vesicular sounds may be heard from day to day gradually extending downward.

The *course* of acute pleurisy is sometimes very rapid ; at other times it may be protracted.

It may terminate in recovery, partial or complete, or it may be fatal in one to two days. At other times it is fatal after a course of several weeks. In the latter cases the patients become very weak and the respiration becomes more difficult, while the mucous membranes become bluish owing to interference with respiration, and animals that have remained in a standing position may now lie or fall down, death following from cardiac failure. In the more favourable cases respiration becomes easier, the pleuritic ridge less marked, the temperature subsides with an occasional rise. The patient commences to feed and is brighter in general appearance and movement.

Chronic Pleurisy.—There are two forms of chronic pleurisy. One is a dry pleurisy, which may be primary in origin, or it may be a sequel to acute pleurisy in which there has been effusion and the fluid has been absorbed. The other form is associated with effusion and may also be a sequel to an acute attack or may develop insidiously. One of the commonest causes of chronic pleurisy in the lower animals is tuberculosis, which is always slow in development. In cattle it is commonly dry pleurisy, while in dogs and cats it is most frequently a moist pleurisy. In the majority of cases in chronic pleurisy adhesions occur between the lung and chest wall and interfere with the full expansion of the lungs. In chronic pleurisy also it is a common thing to meet with recurrent attacks of acute pleurisy.

Symptoms.—In mild cases of chronic pleurisy symptoms are insufficient on which to base a diagnosis, and its presence may actually be unsuspected. This is a matter of importance in the examination of horses as to soundness, as limited areas of chronic pleurisy may be undetected. Symptoms vary according to the character (whether dry or exudative) and extent of the adhesions. There is usually general loss of condition and a dry, harsh, staring coat and intermittent indisposition. In cases where there is considerable effusion the same physical characteristics as revealed by percussion and auscultation are produced as in acute pleurisy. In a dry pleurisy with adhesions respiration is interfered with, especially on exertion, which is likely to produce fits of coughing. Horses with pleuritic adhesions frequently emit a sharp painful grunt when "bulled," *i.e.* placed against a wall and held short by the head and suddenly threatened with a stick. Chronic pleurisy may terminate fatally as the result of exhaustion, while in other cases it may apparently cause very little inconvenience and is not even suspected until after death and a post-mortem examination is made.

Differential Diagnosis.—In the early stages it is not always easy to distinguish pleurisy from other painful conditions of the chest. **Rheumatism** of the chest wall (pleurodynia) is exceedingly painful, but can be distinguished from pleurisy by the absence of general symptoms and on auscultation by an absence of friction sounds. Direct **injuries to the ribs,** in addition to being painful, are generally associated with local swellings and contusions, and in such cases there is usually the additional history of some violence. In **pneumonia** there may be some difficulty in respiration as in pleurisy, but the condition is rarely as painful. Moreover, on auscultation in pneumonia there may be consolidation with loss of sound, but not following a horizontal line as in pleurisy with effusion. In the latter case the heart-sounds are also obscured, while that is rarely the case in pneumonia. An exploratory puncture of the chest wall will usually serve to distinguish. This should be carried out about the seventh intercostal space (about a hand's-breadth behind the point of the elbow in the horse and ox). It may also be necessary to distinguish pleurisy from **pericarditis.** In both conditions friction sounds may be heard on auscultation, but in pleurisy they are synchronous with the respiratory movements, and in pericarditis they are synchronous with the heart-beats. In **hydrothorax** the condition is rarely a painful one, and generally occurs as a secondary affection and associated with general dropsy, which may be of cardiac or renal origin. Symptoms of these conditions then may also be observed. Assistance may also be afforded by tapping the chest and examining the character of the liquid. In hydrothorax the fluid is usually clear and present on both sides of the chest, while in pleurisy the fluid is turbid or contains flakes of fibrin and is frequently unilateral.

Treatment of Acute Pleurisy.—In the first place provide as good hygienic conditions as possible, with reasonable warmth and the application of clothing and plenty of fresh air. If the condition is very painful, relief should be afforded by the hypodermic injection of morphia or the administration of chloral hydrate or the use of salicylates. If the heart is in good condition aconite will also frequently be found useful, particularly in the horse. In the early stages phlebotomy or bleeding may be resorted to, relieving vascular tension and pain. It is only of service, however, in the very early stages. Relief may also be obtained by external applications to the chest wall. In the early stages cold packs may be applied over the painful areas. They allay the sensitiveness and assist in reducing the temperature. Alternatively, and this applies in the majority of cases, hot cloths should be applied to the chest wall, as they afford considerable relief of pain, and since they dilate large superficial areas they afford relief of the internal congestion and check effusion. The application of counter-irritants is frequently recommended, such as a mild mustard plaster or camphor liniment, or a weak cantharides blister. The bowels and kidneys should be kept active with laxatives and diuretics, and for this purpose moderate doses of salines, as sulphate of magnesia (℥ii.-iv.) and potassium nitrate (ʒii.-iv.), may be administered to horses and cattle in the drinking water. In cases of high fever salicylates or antifebrin will be found very useful combined with digitalis and nux vomica to control the cardiac depressant effect. In addition similar medicinal measures may be adopted as have been recommended for the treatment of acute pneumonia, potassium iodide being especially indicated in cases with effusion, since it promotes its absorption and reduces thirst and the intake of water.

Puncture of the chest should be practised as early as possible in pleurisy. It is a mistake to wait until a considerable quantity of effusion has accumulated, and if carried out with antiseptic precautions it is entirely free from danger. In the majority of cases the fluid escapes freely through the cannula. The whole ot the liquid present may be drawn off without any danger. The quantity is sometimes enormous and extends from a few ounces in the smaller animals to 7 or 8 gallons in the horse. The writer has never seen any ill-effects following the removal of these large quantities. In the human subject it is stated that temporary collapse may sometimes follow the removal of large quantities of liquid. To compensate for this, some physicians will actually inject filtered air into the chest. This procedure is stated to have the additional advantage of keeping the pleuræ apart and reducing the formation of adhesions. On the other hand, it is said by some physicians to prolong the course of the illness. Whatever value it may have, however, in human medicine, it is quite unnecessary in veterinary medicine, but the practice in man serves to emphasize the fact that there is no danger of allowing a quantity of air to pass into the chest during the operation. The tapping should be repeated as frequently as necessary. It may be done daily without any ill-effect. Many veterinary surgeons object to tapping in pleurisy, as they say that in their experience, particularly in horses, it has not been justified by results. It is more than probable that in many of those cases the operation was too long delayed for the full benefit of the tapping to be produced. Early and frequent tappings are essential to good results. The operation should be performed about the seventh intercostal space—about a hand's-breadth behind the point of the elbow in horses and cattle. The direction of the puncture should be slightly forwards. It is rarely necessary to use an aspirator, but occasionally the mouth of the cannula requires to be freed from lymph by re-inserting the trocar. When the fluid has been removed it is good practice to inject into the thorax adrenalin hydrochloride (1-1000) ʒi.-ii. in the case of large animals and mm. x.-xv. in the case of the smaller animals. It causes constriction of the vessels and checks effusion, and is at the same time a cardiac stimulant. In empyema of the chest the thick purulent exudate will rarely flow down the cannula; it is then necessary to make a large incision between the ribs to enable the pus-like exudate to be removed. The cavity of the chest should then be irrigated daily with a mild disinfectant. The writer has found warm solutions of hypochlorous acid (eusol) very serviceable in the case of the horse. It is sometimes recommended that a portion of a rib should be resected, but this appears to be unnecessary.

The dietary in acute pleurisy should follow the same lines as recommended in acute pneumonia.

In **chronic pleurisy** with effusion tapping must also be resorted to and repeated from time to time. In those cases of dry chronic pleurisy with adhesions potassium iodide may be administered with a view to reducing the connective tissue, or a course of injections of thiosinamine preparations, such as fibrolysin (horses ʒi.-ii., dog m. x.-xxx.) or iodolysin, may be found of value. In cases of chronic pleurisy any violent exertion should be avoided.

Dosage.—See pp. 544-6.

Hydrothorax

This is a dropsical condition of the pleural sac, and is not very commonly met with in the lower animals. It is an œdema brought about by some venous obstruction and may result from chronic disease of the lungs or of the right side of the heart. Hydrothorax may also occur as the result of disease of the kidney, and in some cases of pronounced anæmia.

Symptoms.—The symptoms and physical changes are those of effusion on both sides of the chest. It is usually painless, but produces very marked dyspnœa with exaggerated thoracic and abdominal respiration, orthopnœa, and rarely any rise of temperature. Coughing occurs on exertion. The effusion may come on quite rapidly in the course of chronic heart disease, and within twenty-four hours the chest may be nearly filled with fluid. On auscultation there is loss of respiratory sounds in the lower part of the chest, but it varies according to the position of the patient, the upper line of the dulness always remaining horizontal. Movement of the liquid may also be heard. This sound may be confused with mucus in the bronchi, but the absence of respiratory sounds in the lower part of the lung will distinguish it. Exploratory puncture establishes the presence of the liquid, which is clear and free from fibrinous flakes.

Treatment.—If the patient is much distressed, tapping of the chest should be practised with antiseptic precautions. This procedure, however, is only palliative and the condition is frequently recurrent. Treatment of the primary condition on which it depends must be resorted to, such as the administration of potassium iodide, digitalis, and nux vomica.

Pneumothorax

This is a condition of air in the pleural sac, but the presence of other gases may be included in the term. The common cause of the condition is wounds of the chest wall involving the ribs and producing direct wounds into the pleural sac. It may also follow an injury to lung tissue, as may occasionally occur in diseases of the lungs. Gas-producing organisms gain access to the chest through these wounds or from an injury or puncture of the œsophagus and produce a similar effect.

Symptoms.—These depend upon the origin of the condition. In cases where there are large wounds of the chest wall there is an extremely severe inspiratory dyspnœa, which comes on acutely. If one side only of the chest is affected collapse of one lung occurs. In the horse there is a considerable danger of the condition becoming bilateral owing to the perforated condition of the mediastinum, in which case death may occur rapidly from asphyxia. The progress of the condition is not so rapid when due to gas-producing organisms. In the latter case the condition is associated with pleurisy, usually of a putrid character. On percussion there is increased resonance over the chest wall of the affected side. On auscultation the respiratory sounds are lost in the pulmonary area on the affected side, but bronchial sounds may still be heard.

Treatment.—When the condition is bilateral and due to large wounds no treatment is practicable, and this is frequently the case in the horse. When unilateral the wound may be closely sutured, the air remaining in the sac becoming eventually absorbed. When the condition is due to gas-producing organisms, the chest must be punctured, and it may be well to make a fairly large incision between the ribs and irrigate the cavity with a mild antiseptic such as eusol. In addition the treatment for pleurisy should be adopted. G. H. W.

Parasites of the Respiratory Tract

Parasites of the Nasal Cavity

Larvæ of the Œstrus ovis. — This parasite is met with in sheep all over the world, and occasionally in goats. It more frequently affects young sheep than adults. The maggots when fully grown are from $\frac{2}{3}$ to $\frac{3}{4}$ of an inch long, and are white or yellowish white in colour, often with dark transverse streaks. They are convex at their dorsal surface, and flat at their ventral aspect, and consist of 9-11 rings which are sometimes striated at the sides. The head possesses two small hooks with which it is attached to the mucous membrane of the nasal chamber.

Life-history.—The adult fly is about half an inch long, resembling somewhat the bot fly of the ox. It possesses a yellow head, brownish-grey thorax, and an abdomen mottled white, yellow, and black. The general effect is to produce a greyish-yellow appearance. The fly deposits its eggs on the nostrils of the sheep in the summer and early autumn. When the flies are about the sheep become very restless and run or huddle together in groups under hedges or in ditches, apparently attempting to hide their noses to prevent the flies depositing their eggs. The eggs hatch out, and the larvæ which issue travel up the nostrils and may become attached to the mucous membrane of the nasal cavities themselves, or they may pass into the frontal sinuses and horn cores or even into the ethmoid bone. The larvæ rarely cause any trouble in these situations until the following spring, when they have attained the size of a horse-bean. They then leave their point of attachment and escape from the nose in the spring and early summer, and are expelled on to the ground. Here they rapidly pass into the pupa stage, and after four or five weeks the adult fly is hatched out and proceeds to attack other sheep.

Symptoms.—The presence of the larvæ causes irritation of the nasal mucous membrane in the spring and early summer. It causes sneezing, nasal discharge which may be unilateral or bilateral, and at first clear, but later becoming purulent and sometimes blood-stained. The sheep shake and toss their heads and rub their noses with their fore limbs and also against hedges and posts, and may actually injure the skin of the face. At the same time there is frequently swelling of the face and a discharge from the eyes. This acute stage generally lasts about a fortnight. In severe cases sheep become very weak and stagger about, and death may follow from exhaustion. In few cases the parasites are said to have penetrated through the ethmoid bone into the cranial cavity, where they set up brain symptoms, such as high-stepping or turning somersaults or going round in circles, thus resembling gid or sturdy.

Treatment.—If the sheep are in good condition when first noticed, it is advisable to slaughter owing to the great loss of condition entailed.

If this plan is not adopted, an attempt may be made to remove the parasites by fumigations with eucalyptus, burning sulphur, or chloroform, and following this with the insufflation of snuff or finely powdered tobacco. This method may be successful if the parasites are in the nasal chamber, but obviously will not displace those in the sinuses or the horn cores, in which case trephining or sawing off the tip of the horn and irrigating with turpentine and soap solution may be practised. In mild affections recovery may be spontaneous, as the parasites tend to leave when they become fully mature. In bad infestations treatment is not likely to be successful.

Preventive measures may be adopted. These consist of applying substances to the nose of sheep which are repellent to the flies, and therefore tend to ward off attacks. A dab of Stockholm tar may be resorted to for this purpose. As this procedure is scarcely practicable in a large flock a special feeding-trough has been recommended, furnished with holes through which the sheep must put their noses in order to get food. The holes are then smeared round with tar and the sheep thus anoint themselves. Mixing a little broom (*Cytisus scoparius*) with trough food is also said to prevent attacks by the fly. Sheep should be kept off pastures known to be infested with *Œstrus* in the day time, and only to be turned out to them at night (see p. 497).

Linguatula tænioides; Pentastomum tænioides; Linguatula denticulata (larvæ).—The adult form of this parasite may be met with in the nasal chamber and frontal sinus or antrum of the dog, and less frequently in the horse, sheep, goat, and cat in Europe, especially Central France. It is a degenerated member of the class *Arachnida*, and is so named from its elongated wormlike body. The sexes are distinct. The male, which is white in colour, is from $\frac{3}{4}$ to 1 inch long, and the female, greyish white or yellow, may be up to 4 or 5 inches in length. The parasites attach themselves to the mucous membrane by means of two curved retractile hooks on each side of the anterior extremity. These may be rudiments of legs. The body may be $\frac{1}{3}$ of an inch broad at its anterior extremity, and tapers off to about $\frac{1}{12}$ of an inch at its posterior end. The skin carries a large number of rings, which produce the appearance as though the worm consisted of a number of separate segments. There is no differentiation between thorax and abdomen. The eggs of the parasite are deposited by the female in the nasal chamber and are expelled by sneezing. They are then ingested by some herbivorous animal, such as cattle, sheep, rabbits, and occasionally horses, cats, and dogs. In the alimentary tract the larvæ hatch out and bore their way through the intestinal wall, and come to rest in the liver or mesenteric glands, or the lungs. In these situations they develop into the pupa stage, and moulting several times become ringed. Later they leave the liver or lungs, and either by way of the intestines or the bronchial tubes may be passed out into the open, or if from the lungs they may become arrested in the nasal chambers, and there develop into the adult stage. Those expelled may become taken in by a second host and reach their predilection seat in the nose. At other times, on the death of the host of the larval stage, infested viscera may be ingested by a dog, who may thus become the host of adult linguatulidæ. The larval stage in the viscera is known as *Linguatula denticulata*.

Symptoms.—The adult parasites in the nose cause considerable irritation and brownish catarrhal discharge which is often unilateral; sneezing; rubbing of the nose with the feet or along the ground or against gate-posts, etc. There is sometimes epistaxis and occasionally dyspnœa. Nervous symptoms may sometimes be set up resembling epilepsy. During fits of sneezing the parasites may be dislodged and expelled from the nostrils, while at other times they are aspirated into the trachea, and may produce fatal results. Positive diagnosis during life may be made by discovery of the eggs of the parasite by the microscopic examination of nasal discharge.

Treatment.—The parasites are not very easily removed from the nasal chamber. They are very resistant to most antiseptics or parasiticides. Nasal douches are sometimes effective, such as weak lysol or weak creolin solutions ($\frac{1}{2}$-1 per cent) or benzine and water. These may be followed or alternated with inhalations as of chloroform, turpentine, or formalin vapour. Finely powdered tobacco may cause the dislodgment of the parasite by setting up increased sneezing. When the parasites are in the frontal sinuses it is necessary to trephine and irrigate with parasiticidal solutions.

Preventive measures consist in moving affected animals off pastures, and preventing nasal discharges contaminating food material of herbivora. Larval stages found in the viscera should be destroyed and not allowed to be eaten by dogs or cats (see p. 800).

Coccidia (p. 222).—In rabbits coccidia, which usually affect the liver and intestines, are occasionally met with in the nose, setting up nasal catarrh and conjunctivitis.

Symptoms.—These consist of sneezing and coughing, with the nasal discharge, salivation, fever, and often difficulty of respiration. Emaciation is also set up. When conjunctivitis is present there is a discharge from the eyes. In rare cases the parasites penetrate up the middle ear and cause otitis, the head being carried to one side and the animal walking round in circles. Diagnosis is confirmed by the discovery of the coccidia microscopically in the nasal discharge.

Treatment.—The same measures must be adopted as for coccidiosis of the liver. As a general rule the best proceeding is to kill off the affected rabbits and exterminate the warren.

Other parasites occasionally found in the nasal chamber are :

Limnotis nilotica (a leech) in the horse, ox, and dog.

The larva of the *Sarcophaga magnifica* (a maggot) in the ox, sheep, pig, and dog.

Parasites of the Larynx

Round worms are sometimes found attached to the mucosa of the larynx in ruminants. They are from ½ inch to 1 inch in length, and red in colour. The sexes are always paired, having a fork appearance. They are closely related to the gape-worms (*S. trachealis*) of poultry, but are rather larger. The species recorded are :

Syngamus laryngeus, affecting cattle in Malay States.

Syngamus nasicola, affecting goats in Tropical Africa.

The symptoms set up are not recorded.

Pulmonary and Bronchial Strongylosis

The principal pulmonary and bronchial strongyles are the following :

All the domesticated animals are subject to infestation of the respiratory tract with a variety of parasites. The most important group are the nematode worms belonging to the Metastrongylidæ, setting up pulmonary strongylosis or lung-worm disease.

Very little is known of the life-history of these parasites except *Dict. filaria.* The adult worms deposit their eggs in the lungs or bronchial tubes, and these or the embryos which issue are carried up into the pharynx, whence some are expelled directly through the nose, while others are swallowed and are expelled with the fæces. It appears probable that some development occurs outside the primary host, but that an intermediate host such as the mollusc, insect, or an earthworm is not necessary. Be that as it may, a certain amount of moisture is necessary, such as is provided by damp pastures, and in these circumstances parasites may remain alive for many months, and are possessed of great vitality. Even under unfavourable conditions, as in dry weather, the parasites may remain alive but more or less dormant until they resume activity again at the onset of moist conditions. The way in which parasites enter their host is also not positively made out, but it is probable that in the majority of cases they are ingested with the herbage, and ultimately reach their predilection seats, where they become sexually mature in from six to eight weeks. The effects of these parasites are most serious in sheep and lambs, and to a less extent in calves and swine. Horses, dogs, and cats are much less frequently and less seriously affected.

Name and Synonyms.	Length.	Habitat.	Host.
Strongylus Filaria (*Dictyocaulus filaria*), white, long, thread-like, ovoviviparous	Male 1-3 ins. Female 2-4 ins.	Trachea and bronchi	Sheep, goat, deer, and camel.
Strongylus rufescens (*Synthetocaulus rufescens*), reddish brown, shorter, oviparous	Male ½-1 in. Female 1-1½ ins.	Bronchioles, alveoli, and lung tissue	Sheep and goats.
Strongylus capillaris (*Synthetocaulus capillaris*) (lung hair-worm), fine as spider's web (thought by some to be undeveloped *Strongylus rufescens*)	As *S. rufescens*	Lung tissue	Sheep and goats.
Strongylus micrurus (*Dictyocaulus viviparus*), long and tapering, white, ovoviviparous	Male 1-2 ins. Female 3-4 ins.	Trachea and bronchi	Calves (horse and ass ?).
Strongylus pulmonalis (*Dictyocaulus pulmonitis*)	Slightly shorter than *S. micrurus*	Lung tissue (vesicles)	In calves (less common).
Strongylus paradoxus (*Metastrongylus apri*), white or brown, ovoviviparous	Male 1 in. Female 2 ins.	Trachea and bronchi	Pig.
Metastrongylus brevivaginatus, very similar to above	As *S. paradoxus*	Trachea and bronchi	Pig.
Strongylus vasorum (*Hæmostrongylus vasorum*)	Male ⅗ in. Female ⅘ in.	Lung tissue and small arteries	Dog.
Strongylus pusillus (*Synthetocaulus abstrusus*), a very short white worm	Male ⅕ in. Female ⅖ in.	Lung tissue	Cat.
Strongylus arnfieldi (*Dictyocaulus arnfieldi*), white, thread-like, ovoviviparous	Male 1 in. Female 2 ins.	Lung tissue	Horse and ass.

Pulmonary and Bronchial Strongylosis in Sheep (*Hoose, Husk*).—The parasites affecting sheep are the *Strongylus filaria*, which sets up the condition of parasitic bronchitis, and the *Strongylus rufescens*, which sets up the condition of parasitic broncho-pneumonia. The former is most commonly met with in lambs, while the latter principally attacks adult sheep, but is not so common as the *Strongylus filaria*.

Symptoms.—As already stated, the symptoms are more serious in lambs than in adult sheep. They consist in most cases of indications of bronchial catarrh with a husky, frequent cough, which is very easily set up when the sheep are disturbed and made to run a few paces. There is usually a thick mucous nasal discharge, in which may be found eggs or embryos and sometimes adult worms. The affected animals, particularly the lambs, rapidly lose condition and become very anæmic. There is rarely much alteration of temperature, and the affected animals frequently rub their noses along the ground or stand with their head depressed, and breathing through the mouth. The appetite is in abeyance, and the bowels are usually irregular. This may be in part due to the frequency with which strongyles are also present in the alimentary tract. The fleece becomes harsh and dry. In bad cases exhaustion, general debility, and dyspnœa are set up, and it may prove fatal from asphyxia in from a fortnight to two or three months according to the severity of the attack. It is usual for a considerable number of animals to be affected at the same time, and the effects are most noticeable in the autumn, especially in wet seasons and on pastures that have been overstocked. Mild attacks in adult sheep may pass unnoticed until post-mortem examination is made in the abattoir.

Anatomical Changes.—In lambs the bronchi are chiefly affected; the bronchial mucous membrane is thickened and may be blood-stained. The bronchi contain a considerable amount of frothy and sticky mucus in which may be found varying numbers of *S. filaria*. In some cases tangled masses of them are curled up together, almost occluding the bronchus. In addition the carcase is usually lacking in fat, anæmic and emaciated.

In adult sheep *Strongylus rufescens* is the offending parasite, and the common lesions are nodules dotted about in the lungs up to the size of a pea. They are immediately below the pleura and often appear at the edges of the lungs. Their colour may be deep red or pale yellowish grey, the latter sometimes being gritty. These nodules are due to the deposition of eggs and the development into embryos in the alveoli of the lungs. This sets up a localized inflammation and the condition is often known as pseudo-tuberculosis of the sheep's lung. In such cases diagnosis may be confirmed by cutting a suspected nodule, squeezing it, and examining a scraping under a low power of the microscope. In some cases where there has been an acute or intensive infestation a pronounced lobular pneumonia is set up in which the red spots are very numerous and distributed throughout the lung.

Treatment.—*Preventive measures* should always be practised in districts where the disease is known to exist. As the young animals are especially liable to serious disease, although old ones may be affected, young sheep should never be pastured after adults. If a fresh pasture is to be used, the lambs should always be grazed upon it first. Pastures known to be infected should be avoided and should be drained, ditches and ponds being fenced off. It should then be dressed with a liberal application of salt and lime. In infested districts it is a good plan where possible to fold the lambs and young sheep on to roots and green crops which are unaffected rather than to risk putting them on to the pastures. The administration of vermicides such as are recommended in curative treatment may also be given as preventive measures to the sheep in infected districts. Experience has proved the efficacy of this practice. The writer knows of flocks in the Fen districts in which for several years in succession when preventive drenching has been practised the loss amongst the lambs has been reduced to very small numbers, but if preventive drenching has been omitted the mortality has again reached a very high figure. It is a matter also for consideration that hay from infected land may possibly convey infection.

Curative Treatment.—In order to be effective curative measures should be adopted in the early stages of the disease, otherwise it is not likely to be very successful. Three methods of treatment have been adopted with varying success.

1. The administration of vermicides by the mouth similar to those used for parasitic gastro-enteritis, particularly turpentine, chloroform, lysol, camphor, benzoin, myrrh, and asafœtida. This method has the additional advantage of destroying gastric parasites, which are so often simultaneously present.

2. Fumigation with various gases. This is often carried out in the treatment of this condition, such agents being used as the fumes of burning sulphur (sulphurous acid gas), chlorine, formalin, tar, turpentine, and the fumes of crude tobacco. For fumigation sheep and lambs are brought into a shed or barn with the ventilators closed, and the fumes are gradually and slowly generated so that the animals get

gradually accustomed to them. Care must be taken to avoid overcrowding or too long exposure to the fumes. A good plan is for an attendant to remain with the sheep as long as he is able to, and on coming out to bring the generating-plant out with him. The sheep may then be left in for a further 10 or 15 minutes, but must be observed periodically during that time. The fumes may debilitate the parasites and by causing sneezing and coughing cause the expulsion of considerable numbers of them. The procedure should be repeated three or four times, with interval of several days or a week.

3. Intratracheal injection of vermicides. This is the most direct method of attacking the parasites. The procedure, however, is not quite so easy in sheep as in calves, and in the writer's opinion the method is not as practicable as the first-named method. The agents used include turpentine, chloroform, creosote, carbolic acid, and iodine solutions. Similar combinations may be employed as are recommended for calves, but employing a quarter of the dose for those animals.

It is of great importance that affected animals should also have a very generous and nutritious diet of cake and corn, and a course of tonics including gentian and iron may be given with the trough food. Sheep and lambs that are very ill are not likely to recover, and should therefore be destroyed and so reduce further infection.

Bronchial Strongylosis (Hoose) in Cattle.—Verminous bronchitis is seen most commonly in calves and young stock up to two years old. Occasionally, but rarely, it attacks adults. The symptoms and course of the disease are very similar to those in sheep. It usually begins with a similar husky cough, becoming very frequent and in paroxysms, but varying according to age and the degree of affection. Young calves may sometimes die of asphyxia within a week, though the course generally lasts for several weeks. There is loss of appetite, wasting, and anæmia, and a more or less copious discharge from the nose containing embryos and parasites and occasionally blood-stained. The temperature is only occasionally elevated 2° or 3° F. Later the weakness becomes more pronounced; the breath may become offensive, and diarrhœa set up. The respiration becomes weaker and more rapid, and the head extended and mouth open. Bad cases may linger from two to three months, and even then terminate fatally. Cattle, however, have considerably more resistance than sheep, and the mortality is correspondingly less.

On *post-mortem examination* bronchitis will be found with occasional patchy pneumonia and some emphysema, and sometimes pseudo-tubercular lesions. The parasites will also be found in considerable numbers, particularly *Strongylus micrurus*, in the trachea and bronchi, and less frequently the *Strongylus pulmonalis* in the lung tissue.

Treatment.—Preventive measures should be adopted in cattle as in the case of sheep. The efficacy of keeping susceptible animals off infected pastures is shown by the fact that stall-fed cattle are very rarely affected.

Curative Measures.—Intratracheal Injections.—As already indicated, this method of treatment is more practicable in cattle than in sheep, and it is undoubtedly the best method of treatment. For purposes of this injection a syringe is required with a very strong hypodermic needle about three inches long. The head of the animal is raised and held as high as possible, and after disinfecting an area in the upper third of the neck on the middle line over the trachea the needle is inserted between the cartilaginous rings. The barrel of the syringe is then applied, and the medicament must be injected *very slowly* so that it trickles down the trachea. If the injection is made too rapidly a violent fit of coughing is set up and asphyxia may result.

There is a wide choice of vermicides which may be used for this purpose, including oil of turpentine, carbolic acid, lysol, oil of cade, oil of cloves, tincture of opium, iodine, and chloroform. These are sometimes made up with a vehicle of olive oil or rape oil, but the writer regards this as a mistake, and prefers to use glycerine. Fixed oils both reduce the penetrating power of the vermicides and increase the difficulty in respiration. The following combinations will be found useful:

(1) ℞ Acid. carbol. liq.
Chloroform āā m. x.
Ol. tereb.
Glycerini āā ʒii.
Fiat injectio.

This may be repeated three times on alternate days.

(2) ℞ Ol. tereb. 100 pts.
Acid. carbol. 5 pts.
Ol. caryophyll. 5 pts.
Tinct. opii 20 pts.
Glycerini 70 pts.
Fiat injectio.

Dose ʒiii.-ʒiv. To be given every second or third day according to the severity of the case.

Vaeth recommends the following mixture:

(3) ℞ Ol. tereb.
Ol. caryophyll. āā 100 pts.
Acid. carbol.
Ol. cadini āā 2 pts.
Fiat injectio.

Dose ʒiiss. for calves.

Scheibel recommends :

(4) ℞ Creosote 1 pt.
Rectified spirits.
Water āā 50 pts.

Scheibel uses this in the form of a very fine spray, which is administered through a special cannula inserted into the trachea.

(5) Dieckerhoff's Solution of Iodine (iodine 1 pt., potassium iodide 5 pts., water 100 pts.) has also been found serviceable. Dose ʒii.-ʒiv.

The administration of *vermicides by the mouth* is frequently resorted to in the British Isles. It is doubtful if agents administered by this channel have any beneficial effect on the worms in hoose. Some of them, however, may be excreted by the lungs and act as pulmonary and bronchial disinfectants. They may assist to some extent in the expulsion of parasites. Moreover, it is a very common thing for animals affected with hoose to be simultaneously affected with gastric strongyles, which materially aggravate the loss of condition and exhaustion. It is claimed that the administration of vermicides by the mouth is particularly efficacious, since both the sets of parasites are attacked. The same parasiticides are employed as have been recommended for sheep: turpentine, carbolic acid, and lysol being the most favoured, and administered either in milk or soap solution.

Fumigation.—This may be carried out in calves as in sheep with such agents as sulphurous acid gas or chlorine. A better plan, however, is fumigation with iodine as follows :

Iodine Treatment for Hoose.—For this procedure one requires two damp sacks, one placed inside the other, and into this is placed a bucket. Next, a hot brick is put into the bucket, the object of which is to volatilize the iodine. When the calf to be fumigated has been secured, a small amount of iodine, which has been previously finely powdered, is placed upon the hot brick by means of a small flat, thin piece of wood. The amount required is approximately as much as could be placed upon a threepenny-piece. Violet fumes are immediately evolved. The mouth of the sack is then held by two attendants and is placed over the nose and mouth of the calf so that the animal inhales the fumes, care being taken that the eyes are not affected. The length of exposure varies with the size and strength of the animal, and one readily learns by experience what the safe exposure is. In an average case one usually waits until the calf has coughed three times. In some cases, however, the calves may cough less readily, and it may be necessary to liberate them after they have coughed once or not at all. The character of the respiration is some guide. It is necessary to apply fresh iodine for each calf, but one hot brick will generally suffice for eight or ten calves. It is therefore necessary to have a number of bricks in the fire heating ready. It is a good plan to have the calves brought up off the pasture and kept without food for several hours before fumigation. The effect on different animals is varied, but it is rare for the fumigation to have any serious ill-effects. Occasionally calves may cough violently and stagger and then become tympanitic, but if they are kept moving about they generally recover after a short time. It is, however, sometimes necessary to puncture them to relieve the tympany. The fumigation may be repeated in ten to fourteen days.

General Treatment.—Affected animals should be brought up off the pasture to avoid further spread of infection and also to reduce additional infection for themselves, and where possible they should be fed in a straw-yard or building. If it is essential that they should be turned out to pasture at all, it should be done later in the day when the dew has gone. Their bodily strength must be maintained with highly nutritious diet with some concentrated food, such as corn or cake, and a course of alimentary tonics such as gentian and iron may with advantage be administered. Animals that are very ill and show no signs of improvement under treatment should be slaughtered in order to reduce infection. The pastures known to be affected should be left free of susceptible animals, but may be pastured by horses or adult cattle. Alternatively they should be dressed with a liberal application of agricultural salt or lime. If the meadow is used for the production of hay the latter should be fed to animals of a different species from those that may have contaminated the pasture.

In Swine.—Parasitic bronchitis in swine is mainly due to the *Strongylus paradoxus*, and as in the case of cattle, when present in small numbers these parasites may give rise to few or no symptoms. In intense infestation, however, they may set up a severe form of bronchitis affecting a considerable number of pigs with symptoms closely resembling those occurring in sheep (spasmodic coughing, nasal discharge, and oral respiration). In other cases there is only slight loss of condition and somewhat increased nasal discharge (snuffles). Positive diagnosis is made by microscopic examination of nasal discharge, or by post-mortem examination of a badly-affected pig, which may be slaughtered for the purpose. In cases where early diagnosis is made and the pigs are in good bodily condition, it is best to send them to the butcher for slaughter.

In horses verminous bronchitis is an exceedingly rare condition. *Strongylus Arnfieldi* occasionally sets up bronchial pneumonia, but its presence is rarely suspected during life. In those rare cases where symptoms are set up they closely resemble those of hoose in calves.

Pulmonary Strongylosis in the Dog.—Small oval cysts have been found under the mucous membrane of the trachea and bronchi in dogs, which on examination are found to contain a small curled nematode worm whose identity is not definitely established. It is probably the same parasite that has been described under the name of *Filaria osleri* and *Strongylus bronchialis canis.* In one outbreak in which they were observed the dogs showed many symptoms closely resembling those of distemper, namely, anorexia, vomiting, catarrh, coughing, general weakness and convulsions. It is highly probable, therefore, that these symptoms were due to distemper and were not set up by the parasite.

Strongylus vasorum.—This parasite may be found in the embryonic stage in the lungs of dogs, having been carried from the right side of the heart and pulmonary artery, where the adult parasite is to be found. The symptoms set up are partly due to the cardiac irregularity and partly to the pulmonary lesions. When present only in small numbers no symptoms may be set up, but when numerous there may be fits of coughing, anæmia, and wasting, and suddenly occurring dyspnœa. The condition rarely terminates fatally. *Treatment* is not very satisfactory ; some observers claim good results from the daily administration of turpentine. Atoxyl or other arsenical preparations may be tried.

In the Cat, *Strongylus pusillus* may set up a lobular pneumonia and cause lesions somewhat similar to those produced by *Strongylus rufescens* in sheep. The symptoms are those of frequent coughing, nasal discharge, sneezing and wasting, often accompanied by vomiting and diarrhœa, and may be fatal in from two to three months. Diagnosis is made by discovery of the ova in the nasal discharge. *Treatment* is very unsatisfactory, and if a positive diagnosis is made probably the best plan is to destroy the animal to prevent further infection.

Echinococcus veterinorum is frequently found in the lungs of horses and cattle and swine, and also in camels. The symptoms set up are somewhat obscure; only when present in large numbers may there be any disturbance of respiration. Rupture of a cyst wall allowing escape of liquid is said to cause skin rashes. It is stated that diagnosis may be made by the complement fixation test and also by the precipitin test.

Treatment.—No satisfactory curative treatment is possible. Preventive measures consist in the administration of worm medicine to dogs and destruction of their excreta.

Distoma hepaticum (see Parasites of Liver).—This parasite is occasionally met with in the posterior lobes of the lung in cattle and sheep, and rarely in the dog. It may give rise to symptoms of broncho-pneumonia, but its presence is rarely suspected during life. In affected animals it is usual to find the liver more extensively involved.

Balharzia (see Parasites of Liver).—The adult parasites are occasionally found in the pulmonary vessels.

Paragonimus westermanni (the Asiatic lung fluke).—This parasite lives normally in cysts in the lung tissue in cats, dogs, pigs and man in the Far East, and in pigs in the United States. It is blood-red in colour, and has the size and shape of a pea. Its presence is only established on post-mortem examination.

G. H. W.

DISEASES OF THE DIAPHRAGM

Spasm of the Diaphragm ; Abdominal Pulsation

This condition may be met with in any of the domesticated animals, but it is seen most frequently in the horse and in the dog.

Etiology.—The cause of spasm of the diaphragm is not clearly understood. In the majority of instances it is thought to be due to reflex action, the result of indigestion or other affection of the gastro-intestinal tract. It may thus be brought about by defective quality of food, such as mouldy hay or corn, and attacks have been brought on by drinking cold water and by greedy feeding. Irritant poisons may also set up spasm of the diaphragm. Spasm of the diaphragm may also be brought about by over-exertion such as may occur in the hunting-field after a fast run, particularly in the early part of the season. Hutyra and Marek record a case in the dog in which the spasms of the diaphragm were due to catarrh of the stomach and bowels. The writer has seen it in a dog occurring at frequent intervals, particularly if any excitement was permitted immediately after feeding. Post-mortem examination in this case revealed that the dog was affected with stenosis of the pylorus followed by dilatation and hypertrophy, and it is reasonable to assume that lesion to have been responsible for the symptoms. Another asserted cause which is worthy of consideration is intermittent pressure or stimulation of the diaphragmatic nerve as it passes through the mediastinum. This can be brought about by enlarged glands in this area and also spasmodic conditions of the heart. In the latter case the abdominal pulsations would occur immediately after the heart-beats, instead of coinciding more or less with the normal frequency of respiratory movements, as is the case when due to other causes.

Symptoms.—These often appear to be very urgent, and may come on very suddenly; they may pass away equally suddenly. The most noticeable feature is a violent throbbing of the whole body generally corresponding with each respiration, although occasionally, as already stated, they may coincide with the heart-beats. Peculiar throbbing action of the abdominal muscles is also noticed, and this can be appreciated most easily by laying the hand flat along the asternal cartilages of the ribs. At the same time there may be observed some apparent bulging of the abdominal muscles in this region, owing to narrowing of the posterior margin of the thoracic wall due to the contraction of the diaphragm. There may also be heard a soft laryngeal sound likened to a sob or hiccough at each respiration. The patient appears somewhat distressed, and in the case of excitable horses profuse perspiration may be set up. During the attack the appetite is in abeyance, and the pulse is generally small and frequent, occasionally palpitating. There is rarely any alteration of temperature. In the majority of cases there are indications of indigestion and irregular condition of the bowels, together with periodical vomiting in animals capable of emesis. The symptoms may pass off in a few minutes, and recur several times in the course of the day; this is frequently the case in dogs. In horses the symptoms are usually more or less continuous for from several hours to several days, and then may suddenly disappear.

Spasm of the diaphragm is rarely serious in effect, and it is doubtful if any case actually proves fatal as the result of this spasm. In cases where death has been recorded, it is more than probable that the fatal result was due to the original cause responsible for the spasm of the diaphragm.

Treatment.—No drastic measures are called for. The spasms during an attack may be cut short by inhalation of amyl nitrite or of chloroform. If that fails hypodermic injection of morphia gr. v.-vi. in the horse or gr. ¼-i. in the dog will cut short the attacks. In the case of animals that are subject to frequent attacks a course of bromide of potassium should be given. During the attacks the patient should be kept quiet and free from excitement. Attention should also be paid to the condition of the alimentary tract, a laxative should be administered such as sulphate of magnesia, and an easily digested diet provided. In addition, in susceptible subjects a period of rest and quietness should be enforced regularly after feeding.

Rupture of the Diaphragm; Hernia of the Diaphragm

This condition in the majority of cases is brought about as the result of a fall or other violence. Occasionally it occurs in the course of colic in horses, and it has also occurred where the horses have been cast for operation, and in that case probably due to violent straining. In dogs it may be due to having been run over.

Anatomical Changes.—The rupture may occur in any part of the diaphragm, but is most commonly met with in the tendinous portion. If the rupture is recent the irregular edges will be more or less blood-stained and congested, with phenomena of inflammation, while in old-standing cases of small ruptures adhesions with the viscera may be produced. Some of the abdominal viscera will usually be found in the thorax, depending on the extent of the rupture. The herniated organs may be portions of the liver, stomach, or intestines. When a small rupture has occurred permitting a loop of bowel to pass through, strangulation is likely to occur with intense congestion and discoloration. In other cases hernia may have occurred through the natural openings of the diaphragm, especially through the œsophageal foramen. In this way loops of bowel of varying length have been found in horses and in cattle; while the writer has observed hernia of half of the liver and gall-bladder in a cat, and of the entire stomach in a dog.

It is important to distinguish ante-mortem rupture from post-mortem rupture of the diaphragm, which not infrequently occurs as the result of gas formation in the rumen of cattle and sheep and of the large intestines of the horse after death. In these cases the rupture is usually very extensive and there is a complete absence of vascular disturbance along the borders of the rupture.

Symptoms.—The symptoms are variable and depend upon the extent of the rupture. If the rupture is small in extent there may be very little to be observed for some time, since the rent may be occluded by some of the abdominal viscera. If, however, a viscus passes through, intermittent attacks of colic will be brought on, and it may ultimately lead to strangulation and death. Dyspnœa is not very marked in small ruptures, but it may be shown in animals going downhill. In slightly larger ruptures of the diaphragm respiratory distress may be observed. The patient stands with his hind limbs partially flexed, and horses occasionally sit on their haunches; they rarely lie down. There may be profuse sweating and a short asthmatical husky cough. In very extensive ruptures intense dyspnœa occurs immediately, and death follows more or less rapidly. In cases where a bowel is passed into the thorax auscultation reveals peristaltic sounds in the chest, while tympanitic sounds may be produced on percussion. In the dog with hernia of the stomach into the chest the most pronounced symptom was persistent vomiting, which could not be allayed by any means, the dog consequently wasted rapidly through inability to retain food. In the cat with the herniated portion of the liver the predominant symptom was wasting, with slight abdominal pain, occasional vomiting, and a yellowish tinge of mucous membranes.

No serviceable *treatment* is available in rupture of the diaphragm.

G. H. W.

DISEASES OF THE NERVOUS SYSTEM

The Brain and its Membranes

Meningitis, or Inflammation of the Membranes of the Brain.—The membranes covering the brain are not infrequently the seat of inflammation in domesticated animals. When the outer membrane, the dura mater, is affected, the condition is called **pachymeningitis.** When the inner membrane, the pia mater, is inflamed, it is called **leptomeningitis.** This is the more common condition, and is much the more serious owing to the intimate association of the pia mater with the brain substance. In fact it is almost impossible for the pia mater to be affected without the brain substance itself being also involved, consequently it is difficult or quite impossible to differentiate between leptomeningitis and encephalitis (inflammation of the brain substance). The third membrane, the arachnoid, which is placed between the dura mater and pia mater, is readily affected by any inflammation which involves either of the other two membranes. Leptomeningitis is usually *general* in distribution; pachymeningitis is frequently *local.* Inflammation of these membranes of the brain may also extend to the corresponding membranes covering the spinal cord, with which they are directly continuous, in which case spinal meningitis is set up in addition to the cerebral meningitis (cerebro-spinal meningitis).

Etiology.—In the vast majority of cases meningitis is brought about by micro-organisms, and in those cases in the lower animals the condition is usually secondary to some specific affection, such as tuberculosis, suppurative diseases, influenza, joint-ill, swine erysipelas, and canine distemper. It may be primary, as in epizoötic cerebro-spinal meningitis. It may be brought about by parasites—*Cœnurus cerebralis* and larvæ of the *Œstrus* in sheep, and *Strongylus vulgaris* in horses. Meningitis may also be due to external injuries, with or without fracture of the skull, while in other cases the infection may take place from an orbital injury or poll-evil in the horse and occasionally from disease of the middle and internal ear. Dietetic errors are also asserted to be causes of meningitis, and this is particularly attributable to excessively nitrogenous foods such as clovers and other leguminous foods.

Symptoms.—The symptoms usually commence by restlessness accompanied by a certain amount of dulness which quickly passes into excitement or delirium, and in rare cases the patient actually becomes aggressive. At this period there is increased sensitiveness or hyperæsthesia. The respirations are accelerated and the pulse quick and frequent, the temperature elevated several degrees. At this stage horses may kick and bite, and even fail to see objects, stumbling when they move about. When cattle are affected, frenzy may occur with stamping of the feet, wild staring eyes, bellowing and becoming aggressive. In tubercular meningitis the affection is slowly progressive and is rarely associated with frenzy. Dogs apparently do not recognize their owners and are very strange in their manner and attempt to hide themselves. If interfered with they are likely to bite, from nervousness rather than aggressiveness. In pigs frenzy may also occur sometimes with exaggerated movements and squeals. At other times they hide themselves under the straw and resent disturbance. In all species this period of frenzy is usually of short duration and is followed by general depression, dulness and listlessness, hanging head, dilated pupils, and rolling gait. Frequently the patient lies down, and can be made to rise with difficulty, and sometimes cannot support itself. When exudate becomes abundant paralysis is set up, varying in degree according to the extent of the affection and the amount of exudate. In some cases there is almost complete paralysis of one side of the body (hemiplegia). The appetite in meningitis is variable. In most cases if the animals retain consciousness they will feed if food is raised to their mouths. They appear to be unable to find it for themselves. In the case of the smaller animals they will frequently masticate and swallow any food that happens to be placed in their mouth. There is usually some degree of constipation and often retention of urine.

In the dog a form of leptomeningitis is a fairly common sequel to distemper, usually making its appearance after the acute stages of that disease have passed. In some instances, in fact, the dog appears to have made a complete recovery before any brain symptoms are observed. The development of the symptoms may then be rapid or protracted. It usually commences with a slight change of demeanour and diminished intelligence, together with a staggering gait. This is usually followed by the dog turning round in circles, which at first are wide, and as the condition advances become smaller until he may ultimately gyrate on his own axis, the head being depressed towards the side to which he turns. By this time there is usually complete blindness with widely dilated pupils and frequently strabismus (squint). At other times one half of the body is more or less paralysed (hemiplegia), and the animal is quite unable to support himself. The writer has also met with cases in which the dog rolled on the ground on the long axis of the body until he arrived at some obstacle which prevented him rolling farther. They generally arrived at this situation on their back, but if turned over and placed on the sternum against the wall they would remain in that position for considerable periods. Sometimes the head becomes depressed and imperfect somersaults are performed; while at other times the reverse action takes place, the head being drawn upwards and backwards, and if the patient's feet are touched a backward somersault results. In these two last cases the cerebellum and its meninges are affected. It, however, frequently happens that the inflammation of the cerebral meninges extends to the spinal meninges, and this generally results in partial or complete paralysis of the hind quarters (paraplegia). When this happens there may be either retention or incontinence of fæces and urine.

In the majority of cases meningitis has a fatal result, particularly in the larger animals and in swine. In dogs their recovery is much more frequent, while in some cases nervous derangements remain as a sequel. In those cases of recovery of the dog, the course is usually a very long one, and may run from two to three months. The writer is of opinion that probably more recoveries would occur if time was only allowed.

Treatment.—In the larger animals treatment is very unsatisfactory, and if due to bacterial causes it is doubtful whether it is worth attempting treatment. In valuable animals, however, it may be policy, as recovery occasionally occurs. When due to dietetic errors the case is very hopeful. Perfect quietness should be provided, and the patient should be left loose in a roomy place so that he is not likely to injure himself. If seen in the early excitant stages venesection should be practised. This will often relieve the symptoms very rapidly. Blood pressure may also be reduced by the administration of a saline purgative. Drastics such as arecoline or pilocarpine have also been recommended, but they are of doubtful value. Nervous irritability may be controlled by the use of potassium bromide, and potassium iodide may be added as an internal antiseptic and also to assist the absorption of fluids from the meninges. Morphia and opium are occasionally given, but owing to their preliminary exciting effects they are contra-indicated. The application of leeches to the head in the early stages may afford relief, and should be followed by cold applications to the poll. When the acute stage is passed a blister may be applied. In cases where there is any paralysis massage of the affected parts or the application of the galvanic battery may restore the nerve current and reduce the muscular atrophy. In the case of dogs in the protracted cases, after the first week or two, nerve stimulants should be given, such as tincture of nux vomica m. ii.-x. alternated weekly or fortnightly with liquor arsenicalis or Donovan's solution in similar doses. If the meningitis is due to traumatic causes a surgical measure such as trephining is necessary, as also in those instances when parasites may be diagnosed, as in gid in sheep.

Dosage.—See pp. 544-6.

Concussion of the Brain.—This condition is produced by mechanical violence affecting the head, and is manifest by modification of brain functions. It frequently occurs as the result of horses running away or being kicked or falling; in dogs, due to being run over, being struck on the head, or falling downstairs or off a table or chair. The effect in many cases is fracture of the cranium with pressure on the meninges and the brain tissue, with hæmorrhage in some cases and œdema in others.

Symptoms.—There is first the history of the injury usually followed by partial or complete loss of consciousness with relaxation of the muscles and short temporary suspension of respiration. In some cases recovery of consciousness is very rapid and symptoms may pass away quickly; in other cases the development of symptoms may be delayed and may not come on for some hours after the injury. In mild cases, on recovery of consciousness the patient may get up and walk away, apparently none the worse for the accident, though vomiting frequently occurs in animals capable of emesis. In more severe cases convulsions may supervene with partial or complete loss of consciousness, or coma may develop. The pupils are usually dilated, respiration irregular, the pulse slow and irregular. The involuntary passage of fæces and urine are frequently observed. In severe cases death may follow more or less rapidly. At other times consciousness gradually and slowly returns after a varying interval, but paralysis of different areas may supervene, which may amount to hemiplegia (paralysis of that side of the body opposite to the side of the head on which the injury has been received). More limited paralyses may occur, such as of a limb or various sets of muscles, or limited to small areas of the head.

Treatment.—Absolute quietness must be provided and a stimulant administered as quickly as possible, such as the cautious inhalation of ammonia; ether or camphorated oil may be administered subcutaneously. The wound may require surgical attendance, and fractured bone may have to be removed or raised. Cold applications to the head are beneficial and tend to check the hæmorrhage. Subsequent hæmorrhage may be reduced by the administration of a saline purgative to reduce blood pressure, and bromides may also be administered in order to keep the patient at rest. In the case of cattle and other food animals it may be advisable to slaughter as soon as possible, owing to the doubtful recovery and the frequency with which some degree of paralysis remains, which may ultimately necessitate slaughter after the carcase has become much reduced in value and possibly rendered unfit for food.

Cerebral Hyperæmia; Congestion of the Brain.—Hyperæmia of the brain may be conveniently studied under two headings, viz. (1) an active or congestive hyperæmia in which there is an increased amount of blood being carried to the brain, and (2) passive or venous hyperæmia, in which there is an increased amount of blood in the brain owing to interference with the outflow.

(1) **Active or Arterial Congestion of the Brain.**—*Causes.*—Arterial congestion may be brought about by anything which decreases the resistance of the arterial walls in the brain, such as loss of tone or fatty degeneration of the arterial walls, or it may be due to derangement of the vaso-nervous centre, resulting in debility of the vaso-constrictors or increased activity of the vaso-dilators. Such derangement may be caused by poisons, either bacterial (distemper), alkaloidal (morphia), or chemical (alcohol); it may also be due to improper foods such as excessive leguminous diet. Active congestion may also be brought about by the direct rays of the sun on the head, as in sunstroke and excessive heat in summer. It may be met with in young animals, both dogs and horses, during dentition. Another cause is increased functional activity either of the brain or of remote organs such as the sexual apparatus. Embolism of certain vessels may cause hyperæmia of the collateral vessels, and in this case the condition is secondary to thrombosis of a large vessel or to verrucose endocarditis (warty growths on the valves of the heart). Active hyperæmia of the brain may also accompany specific diseases such as rabies, and various septicæmias such as swine erysipelas, anthrax, etc. Parasites in the alimentary tract (ascarides) may also cause the condition in dogs and swine and more rarely in horses.

Symptoms.—A variable degree of excitement is the most prominent feature in this condition. On account of the exaggerated movements produced the condition is often called "frenzy" or in horses "mad staggers." Exaggerated movements usually develop rapidly, with restlessness, loss of general voluntary control, and erratic movements. The pupils are dilated and the eyes staring in appearance. Horses frequently plunge forward, knocking into any obstacles apparently without seeing them and often injuring themselves. At the same time there appears to be increased sensitiveness to sound and touch. In cattle similar excitement occurs, with twitching, grinding of the teeth, and frenzy, butting into things with their horns. In dogs there is excitement and restlessness, howling or screaming often with uncontrollable movements and convulsions. In most animals the head appears to be hotter than normal. The pulse is full, quick, and frequent, except when due to intoxications, when it is soft and sluggish. The respirations are accelerated, but largely depend on the extent to which the animal has knocked himself about. The appetite is primarily in abeyance or may be morbid. The temperature varies considerably, sometimes elevated and other times there is no change of temperature. These symptoms may pass off quickly or they may last for hours and may ultimately pass on to encephalitis, of which it is in many instances only the preliminary stage. When this occurs the frenzied symptoms abate and general depression follows. In other cases the acute hyperæmia may result in rupture of a blood-vessel and terminate fatally from apoplexy. When due to intestinal worms the attacks may be very severe and of comparatively short duration, but are often frequently recurrent until the parasites have been expelled.

Treatment.—In the larger animals in acute cases immediate relief to the congested brain may be afforded by blood-letting, but this procedure can only be recommended in the early stages. Relief of the congestion may also be provided by the application of ice or cold packs to the head, the application of clothing, rugs, and bandages to stimulate the superficial circulation, and the administration of a purgative such as aloes to the horse and salines to ruminants and small animals. Frenzy is best controlled by potassium or strontium bromide, which may have to be administered per rectum in doses of ℥i.-iss. for horses and cattle, and ʒss.-i. for dogs. Opium and morphia are contra-indicated for the larger animals on account of their primary stimulant stage; but in dogs, morphia will be found of great service in spite of this possible defect. The patient must be kept as quiet as possible in a dark box, padded if possible. The diet should be sparing in quantity and laxative in character, not too highly nitrogenous, leguminous foods being avoided. Where the cause of the condition is discovered special treatment must be adopted accordingly.

(2) **Venous Hyperæmia; Passive Congestion**

of the Brain.—This condition may be brought about by any obstruction to the return of blood from the brain or anything which diminishes the force of the blood-flow to the brain,—pressure on the jugular veins by tightly fitting collars, or tumours and enlarged thyroids, or valvular disease of the right side of the heart, debilitating disease of the left side of the heart as in dilatation and fatty degeneration. A similar condition may also be set up by reflex action through the pneumogastric nerve in indigestion, and producing a slowing down of the heart's action. In horses this may be brought about by feeding on old, dry, innutritious grasses, as the result of which the terms *grass-staggers* and *stomach-staggers* have been applied.

Symptoms.—The chief feature of venous hyperæmia of the brain is great dulness and depression, usually developing slowly. In rare cases, however, in the horse, there may be a rash involuntary movement, the horse plunging and falling, but when down he is easily controlled. There is dilatation of the pupils, one often being more dilated than the other. When down, partial loss of consciousness may come on, but usually lasts only a short time. The pulse is soft and slow, and the respiration blowing, mucous membrane frequently bluish in tinge. When occurring in harness horses, if due to pressure of the collar, relief speedily follows removal of the pressure, and if the horse has fallen he usually rises after a very short interval. When due to dietetic causes, as is often the case in the horse, the general symptoms of indigestion and pendulous abdomen are coexistent.

Treatment.—If the animal has fallen the head should be raised and any pressure should be removed; a douche of cold water over the head often produces good results. Failing this, immediate relief may be afforded by blood-letting. This should be followed by stimulants such as ether, camphor, strychnine, or caffeine hypodermically, or small doses of atropine. The cautious inhalation of ammonia is also useful. Flicking with the whip or a wet towel stimulates the vaso-motor system and is often productive of good results. Relief of the urgent symptoms should be followed by a laxative, as a large dose of linseed oil or a saline, and a course of nerve and cardiac stimulants such as digitalis and nux vomica should be prescribed. When the condition is due to tumours surgical measures must be adopted.

Special attention must be paid to the diet, which must be as far as possible free from indigestible fibre, and should be slightly laxative in character. Highly nitrogenous foods, as peas, beans, and the clovers (trifolium, sainfoin, lucerne), should be avoided.

Cerebral Anæmia.—In anæmia of the brain there is a deficiency of blood passing through the vessels of the brain. The condition may be brought about by a weak heart, by dilatation of large areas of the body, or by excessive hæmorrhage. It may also be brought about by tumours pressing upon the carotid artery. It is sometimes asserted that the drawing off of liquids from the large body cavities, as in dropsy or in pleurisy, may bring about cerebral anæmia, but although this is not infrequent in the human subject it is extremely rare in the lower animals. In the writer's experience of removing very large quantities of liquids from both the abdominal and thoracic cavities he has only met with one instance in which anæmia of the brain was produced; that occurred in a dog that was already in a state of extreme debility. Cerebral anæmia may also be brought about by a sudden nervous shock or fright. This, again, is very rare in animals, although fairly common in the human subject. The writer has only met with two such cases, and those occurred in dogs. A form of chronic anæmia of the brain may also occur in general anæmia due to specific causes, as in pernicious anæmia.

Symptoms.—In acute anæmia of the brain there is immediate fainting and loss of consciousness; the animal staggers, pigs and dogs vomit, and horses may yawn; control of movements is lost, the animal falls and becomes completely unconscious. The mucous membranes are very pale; the pupils are dilated; the pulse is small and frequent and sometimes almost imperceptible; respirations are usually shallow and irregular, but occasionally slow and deep. Unconsciousness may last a varying period, but usually the symptoms pass away fairly quickly—consciousness returning, but the patient appearing a little dazed. Sometimes dulness and depression remain for several days. It is, however, rarely fatal, but in fatal cases death is usually preceded by convulsions.

In chronic anæmia of the brain general dulness is the predominant feature, the patient is usually very nervous and easily frightened, and convulsions are set up with slight provocation.

Treatment.—Remove any restraint to the neck and throat, and as quickly as possible administer a stimulant such as inhalations of amyl nitrite or ammonia, hypodermic injections of ether, atropine, strychnine, or caffeine. Alcohol, particularly brandy, is also very useful. Flicking with the whip or wet towel is of great therapeutic value. Artificial respiration may be necessary. The cause of the condition must then be diagnosed and treated. If it is due to hæmorrhage considerable quantities of normal saline solution should be injected subcutaneously or per rectum. If due to cardiac or nervous debility a combination of strychnine, digitalis, and phosphates may be given; arsenical preparations will also be found very useful. In cases of chronic anæmia phosphate of iron as in compound syrup of phosphates, together with cod-liver oil and malt, should be given.

Dosage.—See tables on pp. 544-6.

Cerebral Hæmorrhage; Apoplexy; Hæmorrhage of the Brain

This condition consists in a rupture of a blood-vessel in the brain causing effusion and pressure on the brain substance. It is most commonly met with in cattle and dogs, and more rarely in sheep and horses. It may be brought about in cases of debilitated vessels and fatty degeneration in connection with hyperæmia of the brain. It may occur as the result of increased blood pressure during violent exertion. It may also be brought about in various blood diseases such as anthrax, purpura hæmorrhagica, and in parasitic affections of the brain. Similar symptoms may result from rapid exudation into the brain substance from other causes, as in thrombosis or embolism. In cows some of the cases of so-called milk-fever are cases of true apoplexy due to rupture of the vessel in the brain by straining during parturition.

Symptoms.—The symptoms depend upon loss of function of the parts controlled by the compressed centres. In the majority of cases they develop very suddenly; the patient generally performs some erratic movement with loss of voluntary control, trembles, and falls to the ground with partial loss of consciousness. Muscular convulsions are then set up and death may supervene quite rapidly. In other cases the development is less rapid, and premonitory symptoms may be observed, and in slowly progressive cases the extension of the hæmorrhage may be traced by different parts becoming involved seriatim. There may be paralysis of the lips followed by ptosis (paralysis of the upper eyelid), dilatation of one pupil, perhaps contraction of the other, or maybe paralysis of the pharynx, producing dysphagia. In these cases the final attack is generally preceded by some exertion. The animal falls to one side or turns in circles and falls, often struggling, before losing consciousness partially or completely. The mucous membranes are usually injected and the pulse weak and slow, while the respiration is blowing. Sometimes in old animals where the condition is due to fatty degeneration of the vessels of the brain, a similar condition may be observed in the iris in the form of a white ring called the *arcus senilis*. These symptoms are followed by paralysis of different areas, as of single muscles or small groups of muscles (monoplegia), such as one side of the face or of one limb; or there may be paralysis of one half of the body (hemiplegia). When the hæmorrhage involves the cranial nerves of one side, the resulting paralysis affects the muscles of the same side. In all other cases the paralysis occurs on the opposite side of the body from the seat of the hæmorrhage in the brain. Paralysis of the optic nerve may be set up with amaurosis. There may be loss of sensation of one half of the body (hemi-anæsthesia). Complete recovery from apoplexy is extremely rare; it is usual for some degree of paralysis to remain.

Treatment.—In the case of food animals, if they are in suitable condition for food, it is well to slaughter them as early as possible before the carcase becomes injured. If treatment is to be adopted relieve blood pressure in the brain as quickly as possible by blood-letting. Provide absolute quietness and keep the head raised. Cold packs such as ice should be applied to the head. Blood pressure may be still further reduced by administration of a saline purgative and diuretic as sulphate of magnesia and potassium nitrate. Stimulants should be avoided unless there is danger of collapse. In those cases which show signs of recovery a course of potassium iodide, to reduce the effusions, and a small dose of nux vomica may be prescribed. Arsenic is also often administered with good results subsequently. Any local paralysis should be treated by massage and the application of galvanic battery.

Dosage.—See tables on pp. 544-6.

Encephalitis; Inflammation of the Brain Substance

Inflammation of the brain substance is rarely met with as a primary and separate affection; it is more often associated with meningitis. Two forms are recognized: (1) diffuse encephalitis; and (2) localized encephalitis, which may be suppurative or non-suppurative.

Diffuse Encephalitis.—This condition may be met with in all domesticated animals, but is most frequently met with in the dog and the horse, and less frequently in cattle and sheep.

Causes.—Encephalitis may be brought about by micro-organisms as in rabies, or it may be secondary to systemic affections such as influenza in the horse and distemper in the dog. Certain food stuffs are asserted to cause encephalitis in herbivora such as lathyrus sativus, excessive quantities of leguminous foods, and mouldy or otherwise damaged food-stuffs. It may also be brought about by poisonous plants such as the narcotics—wild poppies and hemlock. Poisoning by lead and phosphorus may also set up inflammation of the brain substance.

Symptoms.—Encephalitis is frequently preceded by a stage of active hyperæmia. Consequently in the early stages there is usually a period of excitement with erratic movements and twitchings of various muscles. These symptoms, however, are followed by dulness and depression, stupor, head depressed or resting on the manger or against any obstacle, pupils dilated, pulse slow and feeble but easily disturbed. The respirations are also deep and slow, and sometimes associated with snoring. The appetite is variable, but only completely lost in very severe cases. The temperature is usually elevated 3° or 4° F., although in those instances not due to bacterial causes the temperature may be normal or even subnormal. There is usually some degree of constipation, and the urine is often retained. Circus movements may be developed, while in other cases horses may move backwards or even rear or fall backwards. There is usually more or less loss of general sensation, and there may be paralysis of different areas. If one side of the brain only is involved hemiplegia will be produced. If both sides are affected there may be paralysis of the whole body. **In dogs** encephalitis is most frequently preceded by distemper and is associated with lepto-meningitis, producing symptoms which are indistinguishable from those set up by that affection. No useful purpose will be served by repeating those symptoms (see pp. 176 and 406).

In cattle the preliminary excitant stage is indicated by bellowing and general frenzy. This is followed by stupor, irregular gait, running into obstacles, rolling eyes and amaurosis. Then collapse occurs, and the condition usually terminates fatally in several days. **In sheep** there are various irregular movements, turning in circles, muscular twitchings and staggering, death following in from twelve to forty-eight hours. **In pigs** frenzy is noticed in the early stages with squealing and dribbling and twitching. This stage is followed by depression, stupor, staggering and coma, and is usually fatal.

Treatment.—Put the patient in a dark cool place and avoid all noise and excitement. If seen in the early stages venesection should be practised, and this may be followed by a saline purgative to reduce blood pressure and diminish effusion. If the animal is lying down, the head should be raised, and in the case of ruminants the patient should be propped up on the brisket to prevent tympanites. Cold packs should be applied to the head and potassium bromide should be administered, and may with advantage be combined with potassium iodide. Opium and chloral hydrate should be avoided. When the acute stage is passed the sedatives should be changed to nerve tonics, a course of strychnine and arsenic being substituted for the bromides. Hypodermic injection of nuclein is often found serviceable, particularly in canines. Attention to the diet is necessary; the patient should be tempted with small quantities of easily digestible foods.

The general principles of the treatment of encephalitis are the same as set out for meningitis. Recovery is very rare, but is more hopeful in the case of the dog than other species. When the condition occurs in food animals, if it is seen early before changes have occurred in the carcase, slaughter is the best procedure.

Localized Encephalitis; Cerebral Abscess.—Cerebral abscess is not a very common condition, but it is more frequently met with in young horses than in adults or members of other species. It is rarely primary in origin. It may be brought about as the result of injuries to the cranium or as an extension from disease of the middle ear, specially in the dog and pig following otorrhœa. It may follow disease of the nasal cavities or the facial sinuses. Abscess of the brain may also result from invasion of the blood-stream with pyogenic organisms from remote parts of the body as in strangles in horses and sometimes pneumonia or pleurisy. It has also been met with following metritis, glanders and endocarditis. A similar condition is produced by parasites such as the larvæ of the *Œstrus* and the *Cœnurus cerebralis* in sheep. Foreign bodies such as needles may also penetrate into the brain from the throat or elsewhere and set up localized encephalitis.

The effect of purulent encephalitis is to cause a softening of the brain tissue and the formation of an abscess. As the result of this certain brain centres undergo destruction, while adjoining areas may be deranged in function owing to pressure from the abscess. If the lesion is rapidly developed, symptoms will be more or less exaggerated, while if the development of the condition is slow, the surrounding brain centres appear to be able to accommodate themselves to a considerable extent and the development of symptoms is then slow or symptoms may even be absent.

Symptoms.—The history of a primary disease or direct injury may be available, and is of considerable assistance in arriving at a diagnosis. In cases where the condition is rapidly developed there may be a primary stage of excitement and frenzy, which soon gives way to dulness and very great depression. The temperature is elevated and remains high but fluctuating. In the state of depression the head is hung very low, may be inclined to one side or the other, and there is frequently blindness of one or both eyes. Epileptiform convulsions may occur with varying frequency, and occasional attacks of frenzy may occur even after the state of depression has supervened upon the primary frenzy. These symptoms are followed by coma, and may terminate fatally in from three days to a week. In slowly progressive cases there may be little observed for some days beyond slight dulness or depression, and this is followed by a tendency to turn in circles. The condition then passes on into semi-coma, extreme debility, then suddenly without warning violence may occur and the condition rapidly prove fatal.

Treatment.—Good results are not likely to be obtained except in parasitic or traumatic cases where the abscess may be localized. In those cases surgical measures are called for and consist in trephining the skull and evacuating the abscess, or removing the parasite. In other cases no form of treatment is likely to be successful.

Sunstroke ; Thermic Fever ; Insolation

This is a condition brought about by the direct action of the sun's rays on the cranium. It constantly occurs in the hot weather in animals at work in the harvest field and in horses on manœuvres and other animals exposed to the sun without means of shelter. In the majority of cases the condition produced is hyperæmia of the brain, while in severe attacks paralysis of certain vital centres is brought about with rapidly fatal results.

Symptoms.—These usually develop rapidly with restlessness, excitement, panting, rapid respiration, profuse perspiration and convulsions, with vomiting in animals capable of emesis. The temperature at first may be normal, but often becomes extremely high, may be up to 107° F. The mucous membranes are brightly injected. The symptoms may pass away very rapidly, and the animal may recover within half an hour if placed under suitable conditions. In other cases it is rapidly fatal, death being preceded by convulsions and due to hæmorrhages in the brain. In cases that recover the patient is extremely depressed for days afterwards.

Treatment.—Place the patient in the shade as quickly as possible, apply cold water quickly to the head as from a hose pipe, or a small animal may be held under the tap. Ice packs may be applied to the head and ice enemata administered. Medicinally, rapid anti-pyretics should be administered such as antipyrin, antifebrin, or phenacetin. Quinine is also useful. When the acute attack has passed a saline purgative should be administered such as sulphate of magnesia, and this should be followed by a course of nerve tonics such as quinine, strychnine and arsenic.

Dosage.—See tables, pp. 544-6.

Heat-Stroke ; Heat Exhaustion

This condition is due to an excessively high temperature and especially associated with physical exertion. It is not necessary for the patient to have been in the direct rays of the sun, as is shown by the fact that it may occur during night-time. It is very common in horses during harvest time and in dogs that have been lying out in the sun. It may also occur in cattle and swine during transport in summer, even though the truck may be covered; similarly in such animals when being driven in herds in very hot weather. The effect is thought to be produced as the result of the action of extreme heat on protoplasm of the brain, bringing about cloudy swelling and causing production of toxic substances which act upon cerebral centres. The toxic substance may be carbonic acid or some leucomaine.

Symptoms.—The attacks usually occur suddenly and are very depressant. There are rapidly progressive debility and flagging at work. The respiration is shallow but frequent, the pulse small and weak and may become imperceptible —something like the so-called running-down pulse. The temperature is usually very high, 107°-108° F. The gait becomes staggering, and dogs usually ramble. As the condition advances the patient falls to the ground and is thrown into muscular convulsions. The attack usually terminates fatally, sometimes very rapidly, and in other cases in from one to three days. In those cases where recovery occurs great depression remains for four or five weeks.

Treatment.—Remove the patient to a cool place as quickly as possible, and treat as recommended for sunstroke. If the patient recovers from the acute attack a course of potassium iodide may be administered for a few days, together with a restricted diet, and this should be followed by a course of nerve tonics, such as strychnine and arsenic.

Lightning Stroke ; Electric Shock

Any animal in the open is liable to be struck by lightning. Horses, cattle and sheep appear to be most frequently affected. Electric shock may occur in towns as the result of contact with live wire, as in the case of broken electric tramway wires. The effects vary with the intensity of the flash and the strength of the current to which they may have been exposed.

Symptoms.—In case of severe shock the animal may be killed immediately as the result of over-stimulation of vital nerve centres. In a less severe shock the animal may only be slightly dazed ; at other times may be rendered unconscious without fatal results. There are frequently injuries to the skin, such as singeing of hair along the track taken by the current; at other times the skin may be actually cut through and the subcutaneous structures severely lacerated ; sometimes there is a diffuse singeing over a wide area. Again it must be observed that in some cases there may be a complete absence of any external lesions. In non-fatal cases it is common for some paralysis to remain: it may be paralysis of an ear or the lips, or muscles of the throat or a limb. At other times there may be complete paralysis of the hind quarters, or sometimes there is loss of sight. These sequels, in non-fatal cases, may necessitate the slaughter of an animal owing to their permanent character. In some mild cases rapid recovery may occur after a temporary loss of consciousness.

Treatment.—The treatment of animals affected either with lightning stroke or electric shock is very similar to that for concussion with unconsciousness. Stimulants should be given, such as strychnine, caffeine, atropine, or camphor, all of which may be given hypodermically. For small paralyses massage may be applied and exercise of the muscles. Care must be taken to avoid bed-sores in the case of an animal that remains prone by providing a good bedding, frequently changing the position of the patient and massaging with a little spirit and water the parts that have recently been in contact with the bedding. If there are any local wounds they must be treated with antiseptics, as carbolic lotion 2½ per cent, etc.

Dosage.—See tables, pp. 544-6.

Post-mortem Appearances.—These are of considerable importance on account of insurance claims. Occasionally there may be a complete absence of any characteristic feature or lesion, but the knowledge of a previous thunderstorm and the absence of other lesions sufficient to cause death may assist one in forming an opinion of the case. As a rule, however, the hair is singed or the skin distinctly burnt with the formation of curious figures or dark-coloured streaks and forked or branching lines indicating the track of the current. Under the skin in these areas there may be signs of bruising and hæmorrhage, and staining of the local tissues. There may also be some hæmorrhage from the mouth and nose. Rigor mortis is very slight, setting in quickly and passing off rapidly so that it may actually pass unobserved. The superficial veins are distended with imperfectly coagulated blood, and the serous membranes show small hæmorrhages. Decomposition sets in very rapidly and the body quickly becomes distended. There is frequently no sign of a struggle.

Hydrocephalus; Dropsy of the Ventricles of the Brain

Hydrocephalus may be divided into **congenital** and **acquired. Congenital hydrocephalus** originates before birth, and results in enormous increase in the size of the head and not infrequently gives rise to difficulties in parturition. The forehead becomes very prominent, and the bones of the skull are separated. The condition is probably due to obstruction to the normal exit of cerebro-spinal fluid from the fourth ventricle. The effect is to produce enormous dilatation of the ventricles of the brain, the overlying brain substance being extremely thin. Most animals so affected are still-born, or if born alive they rarely survive longer than a day or so.

Acquired hydrocephalus may be divided into **acute** and **chronic.** Acute hydrocephalus is generally due to meningitis, while the chronic form is due to an obstruction of the aqueduct of Sylvius, which may be brought about as the result of inflammatory exudate, or may be the result of occlusion by pressure by parasites or tumours.

Symptoms.—The symptoms are brought about as the result of pressure of the fluid on the brain centres, and are mainly associated with disturbances of consciousness and movement. The general depression which is set up has given rise in a horse to the name "dummy" being applied to this affection. The patient appears to be sleepy and indifferent to its surroundings, the head is depressed, the eyelids are partly closed and the animal takes little or no notice of any commands or sounds. The position of the legs is unnatural; they are frequently crossed or placed one in front of the other, or one foot resting on another. The pulse is soft and slow, and may be reduced to 20 or 30 beats per minute. Breathing is also deeper and slower than normal. The appetite is variable; the animal may take up a little food and commence to masticate it, and then appears to forget that it is there; and when attempting to drink, the head is often plunged deeply into the water, the animal sometimes appearing as though he were trying to eat it. When an attempt is made to move the horse, he may stagger to one side or the other, but rarely walks straight and puts his feet down as though he were uncertain of the movement. It is almost impossible to get him to back. General sensibility is reduced and the skin reflexes are missing. During the course of the affection sudden erratic movements may also develop with fits of excitement and frenzy. This form is more likely to occur when the condition is secondary to meningitis and is more likely to prove fatal.

The course of acquired hydrocephalus is usually chronic and accompanied by many fluctuations. In some cases the animal may live for months or years, but is worse in the warm weather. It comes on very slowly and gradually, but is rarely recognized in heavy draft-horses until it is well advanced; while in riding-horses it is usually recognized much earlier. In the majority of cases the bodily condition is maintained fairly well in spite of the small amount of food taken, but owing to the general disturbance of motion and the unfavourable prognosis, affected animals have to be eventually destroyed. In other cases the animal may actually die of starvation.

In other species than the horse somewhat similar symptoms are developed, mainly associated with depression and stupidity, and disturbance of vision and hearing; eyes dilated and head even held obliquely and perhaps pressed into a corner. It is seen not infrequently in dogs associated with meningitis after distemper.

Treatment.—In the horse treatment is apparently useless. In the early stages the animal may remain useful for some time for slow work. The administration of pilocarpine grains ii.-v. or arecoline grains i.-ii. afford relief, which may be maintained by the daily administration of sulphate of magnesia ℥iv. In dogs a smart purgative should be administered and repeated weekly, and a course of iodide of potassium prescribed. In dogs lumbar puncture may also be of service.

In acute cases the same treatment should be adopted as for acute meningitis.

Tumours of the Brain

In the lower animals brain tumours are comparatively rare. They are met with, however, more frequently in the horse and dog than in other animals. The commonest tumours are psammomata, which develop in connection with the choroid plexus in the lateral ventricles or in the fourth ventricle under the cerebellum. Cholesteatomata are also met with in the brain substance. Other tumours occasionally met with are glioma, sarcoma and melanoma, and rarely carcinoma. Other tumours found in the cranium are generally in association with the meninges, such as fibroma, lipoma, sarcoma, and carcinoma. Occasionally tuberculous growths and actinomycotic lesions are met with and produce similar effects.

Symptoms.—The symptoms are extremely variable, but owing to their slow mode of growth the tumours may attain considerable size without giving rise to any symptoms. In the majority of cases, however, symptoms are gradually developed and are produced by pressure on the brain substance which may interfere with intelligence and also with voluntary action. Sometimes the first noticeable change consists of erratic movements, giddiness or walking in circles, or leaning the head up against a wall. The pupils may be dilated, or one may be dilated and the other contracted. Sometimes frenzy is suddenly developed; the horse may knock himself about, and may even try to climb the wall. Between the attacks of frenzy the patient may appear comparatively well except for slight depression. At other times the depression becomes very intense and passing into semi-coma. According to the position of the tumour paralysis of certain nerves may be set up. There may be monoplegia of the facial muscles or of the lingual muscles; and at other times hemiplegia (paralysis of one half of the body). In the dog paralysis of the facial nerve and optic nerve has been observed, sometimes associated with ataxia or staggering gait.

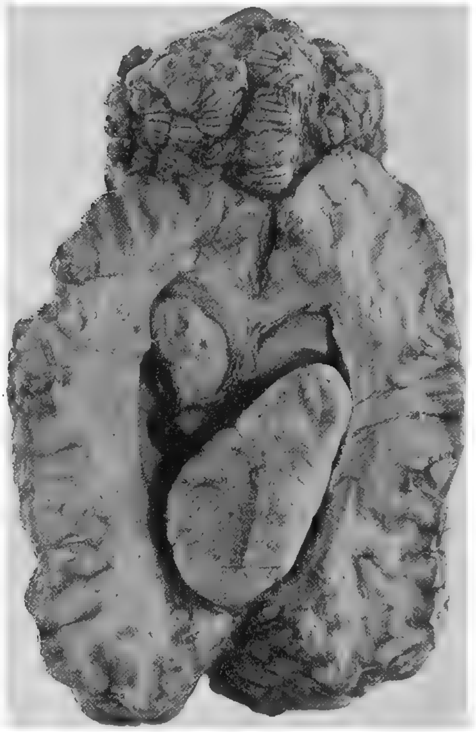


Fig. 79.—Psammoma in the lateral ventricle of the brain of a horse.

Treatment.—Treatment is quite hopeless, and if diagnosis is positive the animal should be slaughtered.

Vertigo; Megrims

Under the heading of Megrims are included conditions associated with more or less suddenly occurring loss of equilibrium and inability to remain standing, and often associated with some degree of loss of consciousness. The condition occurs most frequently in horses and dogs. In horses it generally occurs when the animal is working, most frequently in harness, though it occasionally occurs in the stable and also in riding-horses. The attacks may be periodical with varying intervals which may extend to months or even years. It is doubtful whether the condition should be regarded as an independent affection. In the view of the writer it is more correctly regarded as a symptom which may occur in a variety of affections.

Etiology.—In the majority of cases megrims results from some affection of the brain such as cerebral congestion, dropsy of the ventricles (hydrocephalus internus), tumours or parasites. Megrims may also occur as the result of meningitis. A vertigo may also occur as the result of interference with sense organs, as the eyes. When due to defective vision attacks are more likely to occur either as the result of moving quickly in a circle or travelling in a railway train in open wagons, the objects passing quickly before their eyes. In this respect sea-sickness and train-sickness are closely allied, vertigo frequently occurring in each and affecting both horses and dogs and less frequently other species. Disease of the middle ear also affects the equilibrium, and may set up staggering and falling as in Menière's disease. The so-called **congestive vertigo** due to disturbance of circulation of the brain is perhaps the commonest form. This is almost always associated with interference with the circulation either as the result of a weak heart (dilatation or incompetence) or to obstruction of the jugular veins by tightly fitting collars or throat lashes. This latter cause is in operation in many of the cases occurring in harness horses, as is shown by the fact that the attack speedily passes away on the relief of pressure and the same animals are frequently unaffected except when in harness. Other attacks of vertigo are undoubtedly reflex from the alimentary canal and may be brought on by improper feeding (grass-staggers) and in some cases probably by intestinal parasites.

Symptoms.—The attacks usually come on when the animal is at work, the horse steadies down in pace, often twitches the ears and shakes the head; the facial muscles may also be observed twitching. He then sways and staggers, and if not speedily stopped will probably fall to the ground, or if in double harness he leans up against the pole. If it occurs in the stable the horse appears at first a little nervous, and twitches and leans up against the wall and then falls. When down consciousness is temporarily lost, the muscles being loose and flaccid and the animal lying perfectly quiet. The eyes roll and the pupils are dilated. Consciousness usually returns fairly rapidly, and the horse may then struggle somewhat, and ultimately rises, appearing more or less dazed. The duration of the attacks rarely exceeds four or five minutes. Vertigo can readily be distinguished from epilepsy by the absence of muscular convulsions and the rigid condition of the limbs such as are met with in epilepsy.

Treatment.—During an attack of vertigo all restraint such as collars and straps should be released and cold douches should be applied to the head, which should be raised. Care should be taken that the animal does not injure himself. An attempt must next be made to diagnose the cause of the condition, which if discovered must be treated accordingly. If due to defective harness, such as tightly fitting collars, the necessary correction must be made, and the attacks will then cease. If due to a defective heart a course of cardiac tonics such as digitalis, strychnine and arsenic may be given and excessive exertion or excitement avoided. When the cause is associated with defective vision the application of blinds is said to have a very beneficial effect and to prevent recurrence. If the condition is reflexly produced from the alimentary tract, a purgative should be administered, to be followed by a course of alimentary tonics as recommended for indigestion, and care must be exercised in the subsequent line of feeding. In confirmed cases of megrims it is doubtful if the animal is worth treating owing to the danger of attacks occurring when the animal is out, and the risks thus involved both to the driver and the general public.

Menière's Disease

This is an affection corresponding with vertigo but brought about by disease of the semicircular canals of the internal ear. It is not a very common condition, but is likely to be confused with megrims in horses. In dogs it is likely to follow bad cases of inflammation of the external ear with perforation of the tympanum and extension to the internal ear. In rare cases it occurs in the dog without any previous affection of the external ear.

Symptoms.—The animal rotates the head towards the affected ear, and has a tendency to walk in circles, and frequently staggers as a rule to the same side, and may fall. On rising he appears to be somewhat dazed. In dogs the symptoms are usually more or less continuous. In the horse in some cases the symptoms are intermittent, while in other cases they are continuous. The condition usually commences as in the dog with some apparent affection of one ear, which may be noticed twitching or hanging a little more to the outside than normal. The head is rotated to that side, and when left alone in the stable the horse will frequently turn in circles to that side and may stagger and fall, but rarely loses consciousness. There is a complete absence of any excitement or other systemic disturbance. In a case recently observed by the writer the symptoms were continuous and in the left ear. The horse walked round in circles to the left side, and when attempts were made to turn him in the opposite direction he threatened to fall. Examination of the external ear failed to reveal any abnormality until the case had been under observation for three weeks, when a slight discharge was noticed. The horse staggered frequently to the left side as though to fall, but only rarely fell. It was found impossible to make the horse back, and when he was forcibly pushed backwards he threatened to fall again to the left side.

Treatment of Menière's disease is practically hopeless.

Epilepsy; Falling Sickness

This is a chronic nervous condition characterized by complete loss of consciousness and muscular convulsions which may be tonic or clonic. In true epilepsy there is some organic disease of the brain substance. The condition is rarely met with in the horse and other domesticated animals except the dog, which is a comparatively common subject of epilepsy.

Etiology.—The actual cause of the condition is not well understood, but heredity must be regarded as an important factor. It is generally regarded as being due to increased irritability of the reflex centres, and this may be hereditary in origin or may be due to toxic effects of impaired metabolism or result from acute fevers. Attacks in affected subjects may be brought about by shocks, mental or physical, or excessive excitement, sexual or otherwise. Between the attacks the patient appears to be in normal health. When the attack lasts only a very short time it is called *petit mal* or an epileptiform fit. When the attack is of long duration and the convulsions more pronounced and the loss of consciousness more prolonged the condition is called *grand mal* or *haut mal*.

Symptoms.—In the majority of cases the patient suddenly falls down without any warning and immediately becomes unconscious; his muscles are thrown into general convulsions, the limbs being stiff and rigid, the muscles of the neck tensely contracted. There is champing of the jaws, and the eyes have a fixed expression and frequently rolling, the pupils being dilated. In some cases before unconsciousness occurs a cry may be emitted by dogs, while cattle are said to bellow in a peculiar manner. Unconsciousness may last a minute or sometimes considerably longer, even up to an hour, and during this spasm the breathing may be spasmodic and intermittent, the pulse hard and small, and the superficial veins may appear prominent. There may be involuntary passage of fæces and urine. When consciousness returns the breathing improves and the pulse becomes normal, but the patient remains in a state of considerable exhaustion. There is seldom any serious result from an attack unless the patient has knocked himself about and produced mechanical injury or bitten his tongue. Frequent attacks will cause great debility, and death may occur as the result of great exhaustion produced. In epileptiform fits (*petit mal*) there is only very slight loss of consciousness, and spasms are limited to small groups of muscles. These fits are very difficult to distinguish from vertigo or megrims.

Prognosis.—Prognosis must be based upon the length of the intervals between the attacks. If the attacks are frequent prognosis is unfavourable. In some cases, however, the intervals between the attacks gradually extend, and they may be regarded in a more favourable light, and the patient may grow out of it, but complete recovery is rarely to be expected. Epilepsy should be distinguished from attacks of convulsions of a reflex character, which may be brought about by peripheral irritation as in the case of intestinal worms in young animals or in metritis. These epileptiform attacks would be better described under the heading of eclampsia since they are not due to any direct brain lesion, and they disappear when the cause has been discovered and removed.

Treatment.—During an attack all restraint should be released, and the patient should be prevented from injuring himself. It is sometimes advisable to put a gag into the mouth to prevent the animal biting his tongue. The spasms may be cut short by inhalation of amyl nitrite, but nothing further can be done until consciousness returns. Of the various agents that have been employed to relieve epilepsy, bromides are by far the most serviceable. Their effect is to reduce irritability of the nerve centres. The form of bromide adopted is not a matter of much importance, it may be bromide of potassium, or sodium, or ammonium or strontium. In the case of the horse bromides must be given in large doses of ℥i. twice daily, and in dogs from gr. xv. to ʒi. may be given twice or three times a day. When the attacks are less frequent a course of nerve tonics may be substituted, such as liquor arsenicalis, silver nitrate, lactate of zinc, nux vomica, or valerian. A course of compound syrup of phosphates is also useful. All excitement and violent exertion should be carefully avoided. The diet throughout should be somewhat laxative in character and moderate in quantity. Since the condition is sometimes hereditary affected animals should not be used for breeding purposes.

Dosage.—See tables on pp. 544-6.

Eclampsia; Reflex Epilepsy

Eclampsia is a condition in which convulsions occur that are very difficult or almost impossible to distinguish from epilepsy but in which there is no organic disease of the brain.

There are general convulsions with sudden loss of consciousness dependent on peripheral irritation. They do not recur after the removal of the irritant. Herein lies the main distinction between eclampsia and true epilepsy, together with the fact that the former is not in any way hereditary, while the latter is frequently transmitted from parent to progeny. Eclampsia is most commonly seen in young animals, particularly dogs and pigs, less frequently in adults. In young animals it may occur during teething, and is also frequently due to intestinal parasites. It may also be dependent upon any other irritation of the alimentary tract.

Symptoms.—There may be some warning of an impending attack in the form of general restlessness and some strangeness of demeanour. The attack then develops rapidly with muscular convulsions and rigid limbs and sudden loss of consciousness, which is frequently not so complete as in true epilepsy, while the muscular convulsions during the attack are not so continuous. The attacks may follow one another very quickly and may terminate fatally, while in other cases death is ultimately brought about as the result of exhaustion.

Treatment.—The attacks can be controlled by the administration of a course of potassium bromide or chloral hydrate, which should be administered several times daily in small doses. The cause of the condition should then be ascertained and treated accordingly. When due to teething a sedative mouth wash should be prescribed, and when due to worms, vermicides should be administered. When the cause of the condition has been removed, there is no recurrence of the convulsions.

Parturient Eclampsia ; Puerperal Convulsions

This is a peculiar affection associated with parturition and met with in the lower animals particularly amongst bitches, and more rarely in other females as the goat and sow, and still more rarely in cows and mares.

In the majority of cases puerperal convulsions occur within a few days after parturition, although they may occur a few days before that act. The actual cause of the condition is not well known. It is thought in some cases to be due to irritation which may arise in the uterus or in the milk glands. By some authorities it is thought to be due to disease of the parathyroids.

Symptoms.—Symptoms usually commence with restlessness, an anxious expression and convulsions with tonic or clonic spasms of the muscles. The limbs become stretched out and more or less rigid. Consciousness, however, is usually retained, in this respect differing from true epilepsy and true eclampsia. The temperature is elevated several degrees. The mucous membranes are injected, pulse and respiration accelerated. The attacks may last several days or only a few hours. As the result of convulsions there is great exhaustion, which may terminate fatally unless the patient be given proper attention.

Treatment.—The best results are obtained by narcotics such as morphia hypodermically, or chloral hydrate, or veronal, and the patient should be kept under the effect of these agents until the spasms have entirely disappeared. On account of the probable association of parathyroid defects with the cause of the condition parathyroidin may be given. The milk glands are rarely active, but if necessary they should be relieved. As a precautionary measure also an antiseptic pessary should be inserted into the uterus.

Chorea ; St. Vitus' Dance ; The Jumps

Chorea is a condition in which voluntary muscles or groups of them are affected with clonic spasms or constant twitchings, and the affect is brought about through defective nerve supply. There is no loss of consciousness, and rarely is there any interference with local sensation. It is by far the most common in young dogs, in which animals it frequently follows an attack of distemper. The condition, however, must not be confused with epileptiform seizures, which may occur during the course of distemper in dogs. As a sequel to distemper, chorea may develop some weeks or even months after apparent recovery, and there is no relationship whatever between the severity of an attack of distemper and the liability to the development of chorea as a sequel. In fact in many cases the distemper may have been so mild as to escape observation, until the owner is questioned and he recalls that a short time previously the dog had been affected with slight catarrh and occasional cough which was apparently insignificant and passed away without treatment.

In the horse choreic symptoms have been observed in cases of brain tumours ; while in other cases similar symptoms have complicated endocarditis and intestinal catarrh. These cases, however, should not be regarded as true chorea.

Symptoms.—The characteristic features of chorea are muscular twitchings which frequently begin about the temporal muscles and the lips, and in the early stages may only be suspected when the hand is placed on the head. The spasms gradually get worse and result in constant nodding or spasmodic shaking. In other cases twitchings may affect one or more limbs, fore or hind, either primarily or associated with head symptoms. Consciousness is not affected, and the appetite is usually maintained.

In the majority of cases in dogs the condition is slowly progressive, and the patient gets no rest and loses condition through muscular exhaustion. In some cases, however, the spasms are much reduced or in abeyance during sleep. In bad cases the condition develops into paraplegia or paralysis of the hind limbs, with relaxation of the sphincters of the anus and bladder, causing involuntary passage of urine and fæces. In other cases urine and fæces are retained owing to the physical inability of expulsion; whimpering or crying is then set up, due to abdominal pain or to fretfulness. In some cases there is developed an obstinate ulceration of the legs following the course of the nerves, and dogs may gnaw the affected parts. This condition appears to be due to defective innervation, and may be regarded as a form of shingles (herpes zoster).

In the **horse** choreic spasms have been observed affecting various parts of the body, such as the lips or ears, or a foot, or a whole limb, or sometimes involving the whole body. In **cattle** high-stepping has been observed even when not in the act of progression; swaying of the head has also been noticed. In young **pigs** the symptoms correspond very closely with those of the dog.

Prognosis is, as a rule, unfavourable. In mild cases the progress of the condition may be checked and recovery sometimes occurs. In the majority of cases, however, the affection is progressive in spite of all treatment, and ultimately proves fatal after a prolonged course from exhaustion and wasting, together with hypostatic congestion of the lungs.

Treatment.—If the twitchings are very marked and continuous, it is best to keep the patient under the influence of potassium bromide, which diminishes the convulsions and reduces the exhaustion. The bowels should be opened with a smart purgative which should be repeated at least once a week. When the convulsions are diminished or not primarily exaggerated nerve tonics may be given; the best results probably being obtained by the combination of nux vomica and potassium iodide to reduce effusions in the spinal cord. Easton's Syrup, valerian, arsenic, silver nitrate and citrate of ammonia and quinine are all found useful. Whichever line of treatment is adopted should be persisted in for at least two to three weeks before a change is made. Quite a good plan is to administer nux vomica or Easton's Syrup for a fortnight at a time, alternated with liquor arsenicalis or Donovan's Solution for a fortnight. Voluntary exercise must be limited; the patient must be kept as quiet as possible, all forms of excitement avoided, but with a plentiful supply of fresh air. The affected limbs may be massaged with stimulating liniments, or the galvanic battery may be applied daily for short periods with a comparatively mild current, care being taken not to exhaust the affected groups of muscles. Considerable relief may be derived from the application of a blister of biniodide of mercury (1–8) to the poll when the head is the principal seat of symptoms, or to the back over the cervical thickening of the spinal cord when the fore limbs are affected, or to the loins when the hind limbs are affected. When skin lesions are present a simple emollient astringent such as zinc ointment should be applied, and the parts protected by bandages when possible. In animals unable to stand, attention must be paid to the condition of the bowels, which may have to be relieved by an enemata, and the condition of the bladder, which if distended can be emptied by pressure through the abdominal wall. Foods should be of a laxative character and not too highly nitrogenous, and the water supply should be somewhat curtailed in order to reduce effusions.

Diseases of the Spinal Cord

Spinal Meningitis; Inflammation of the Membranes of the Spinal Cord

Inflammation of the spinal meninges alone is a very rare condition. It is much more frequently associated with cerebral meningitis (cerebro-spinal meningitis), and in the majority of cases the spinal cord itself is also involved owing to its very close association with the pia mater (meningo-myelitis). Spinal meningitis is most frequently met with in the dog and in the horse, next in cattle and more rarely in other species. In the majority of cases it is secondary to some contagious disease such as influenza, strangles and cervical tuberculosis in the horse, distemper in the dog, and tuberculosis in cattle and swine. It may also be brought about by injuries to the spinal vertebræ, as in broken back, and by extension from poll-evil and fistulous withers. In the dog it may be the result of being run over.

Symptoms.—Symptoms are generally ushered in by general stiffness of the back and alteration of gait. The back is usually arched, and there is often increased sensation of the skin of the back in the early stages and evidence of pain on palpation, and there may be spasms of the muscles of the back and of the limbs. Dogs with this condition will frequently cry out in pain when they are lying unmolested. The respiration is hurried and the abdomen becomes tucked up. There is loss of appetite and frequently retention of urine and fæces, owing to the pain produced by the muscular contraction necessitated for their expulsion. As effusion between the membranes occurs the hyperæsthesia passes away and local anæsthesia supervenes. At the same time the motor nerves have become compressed and partial paralysis is set up, usually involving both hind limbs. This causes a rolling gait and staggering with intermittent convulsive movements of the limbs. When the effusion becomes still more copious complete paralysis and loss of sensation of both hind limbs is set up (paraplegia), the patient being no longer able to stand. Dogs may appear fairly bright in manner and attempt to run about on their fore legs dragging their hind limbs along. In many cases where this occurs myelitis has also been set up.

In the case of the larger animals with paraplegia bed-sores develop somewhat rapidly, but bed-sores are not common in dogs affected. Retention of urine and fæces frequently also occurs in this stage owing to the paralysis, and is frequently responsible for whimpering or fretful crying on the part of dogs. In other cases there is involuntary passage of both urine and fæces.

The course of the condition varies considerably. In horses it is comparatively short and may terminate fatally in a few days to a week. In dogs it is frequently protracted, and although it may prove fatal in some cases within a week or ten days, it more frequently lasts several months and even then may terminate in recovery.

Treatment.—The patient should be placed in the best hygienic conditions procurable with a good bedding. Absolute quietness should be observed, and the stable or room should be in a state of semi-darkness. In the case of larger animals if they are still able to support their weight, but not otherwise, they should be placed in slings. A smart purgative should be administered, preferably a saline, and this should be followed with a course of salicylates, potassium iodide, and potassium bromide in the early painful stages. In the subsequent painless stage the bromide should be discontinued and nux vomica substituted. Quinine and arsenic will also be found useful. External treatment may be adopted. In the early painful stages cold applications such as ice packs may be applied to the back. This may be followed in the subsequent painless stage by the application of a stimulating liniment (lin. camph. ammon. 1 pt., lin. saponis co. 8 pts.) or a mild blister (biniodide of mercury ointment 1-8). The writer constantly uses this latter application for dogs, with apparent good results. He has never observed any ill-effects, except in one case in which the dog licked off the ointment, thus setting up gastro-enteritis. In the majority of cases, however, the patient will not attempt to lick it off. Attention must be paid to the removal of urine and fæces in cases of retention. In the larger animals it may be necessary to pass the catheter, while in the smaller patients compression through the abdominal wall usually suffices. Retention of fæces must be controlled by enemata. Feeding is also a matter of some importance. In the majority of cases, except in the early painful stage, the patient will feed voluntarily, foods of a laxative character being selected. If inappetence exists artificial feeding must be resorted to.

Epizoötic Cerebro-Spinal Meningitis in Horses

(See p. 46)

Ossifying Spinal Meningitis in Dogs; Chronic Ossifying Pachymeningitis

A peculiar condition of pachymeningitis is occasionally met with in dogs, associated with a formation of small plates or spicules of bone in the dura mater, causing pressure on the spinal nerves or even on the cord itself. The cause of the condition is quite unknown. It is thought to be of rheumatic origin, but the writer has seen it in cases where there has been no previous history of rheumatism and where the condition has been apparently primary. Age and sex appear to have no influence on the occurrence of the affection. All the cases seen by the writer have occurred in small dogs, while Hutyra and Marek state that it appears more commonly in the larger breeds. Gray states that the condition may be hereditary.

Symptoms.—The condition is slow and insidious in onset, and is first noticed by pain evidenced by the dog such as crying out without any apparent cause on movement or on being handled. Any movement is carried out very cautiously, and the whole body is rendered as stiff as possible. The patient objects to the head or neck being moved from side to side, or being raised or depressed. The head is often extended, and when an attempt is made to depress it forcibly the neck and body are held so rigid that the dog will actually pivot on his fore limbs, his hind legs being raised from the ground. In some cases progression may actually take place on the fore legs only.

The severity of the symptoms fluctuates; there may be intervals during which the patient appears to be quite normal. Affected animals usually feed fairly well throughout the course, but in spite of that fact there is considerable wasting and loss of condition.

Treatment.—The treatment of this condition is very discouraging; and although temporary improvement may be obtained the majority of cases prove fatal. A purgative should first be given, which may be assisted by enemata.

This should be followed by analgesics, the salicylates affording the best results, acetosalicylic acid or sodium salicylate. Potassium iodide and quinine may also be useful. Unless there is some improvement within a month or six weeks it is rarely advisable to continue treatment.

Spinal Concussion; Concussion of the Spinal Cord; Injuries to the Spinal Cord

The most common cause of this condition in the horse is injury occurring in the region of the neck or the back, with dislocation or fracture of the vertebræ, as the result of a fall, most frequently in the hunting field. It may also occur as the result of violent muscular contraction when a horse has been cast for surgical purposes, as in old horses with ankylosed spine, and especially if the head has not been kept fully extended. The injuries to the bone involve damage to the spinal meninges, and often to the spinal cord itself, and are generally associated with some degree of hæmorrhage. Fractured spine is comparatively uncommon in cattle, and after the horse it is most common in dogs as the result of being run over. Injuries to the spinal cord have also occurred as the result of penetration of the spinal canal with sharp bodies such as a darning needle.

Symptoms.—Symptoms vary according to the position of the injury. If a severe injury occurs in the upper half of the neck, death is likely to occur almost immediately owing to paralysis of respiration. At the same time the whole body and limbs are paralysed, movement only occurring in the muscles of the head. When the injury occurs posterior to the cervical vertebræ, paralysis of the hind half of the body takes place. There is loss of sensation over the same area, as indicated by lack of response to pricking with a pin. There is often profuse perspiration of the paralysed portion of the body with a sharp line of demarcation between it and the unaffected anterior areas. There may be either incontinence or retention of urine and fæces. There may also be subcutaneous swelling and contusion marking the seat of the injury, and crepitus may be felt on forcible movement of the spine by lifting the tail. If the injury affects only one-half of the spinal cord, paralysis of the muscles occur on the same side, and there is also loss of sensation on the opposite side posterior to the injury.

In the case of dogs that have been run over there is a similar paralysis posterior to the injury. Crepitation may be felt on manipulation, and there is usually a local swelling and discoloration owing to the bruising of the tissues.

Fracture of the spine may occur with deferred displacement and consequently delayed injury to the spinal cord in both horses and dogs. The writer has met with a number of cases in the horse in which the animal has fallen sometimes in jumping short across a ditch, after which he has walked varying distances, a mile or so to his stable. Subsequently, frequently during the next night, the animal has lain down but has been unable to rise as the result of a broken back. Such cases are probably fracture with deferred displacement. Symptoms of concussion may also be deferred in the case of a dog. The writer recalls an instance in which a fox terrier was run over by a van and was able to walk about for a week before paralysis of the hind limbs set in. The paralysis lasted seven or eight weeks and the animal ultimately made a complete recovery.

Treatment.—In cases of spinal concussion, associated with fracture of the vertebræ, treatment is practically hopeless and affected animals should be slaughtered. In other instances, however, where there is no fracture, treatment may be undertaken. Perfect quietness should be prescribed and a good bed provided. Massage of the back and limbs should be practised, and in the later stages the electric current may be applied. A course of bromides should be given for the first week or two, and should then be followed with nerve tonics such as nux vomica and arsenic. Iodide of potassium is also serviceable in reducing effusions in the spinal canal. The bowels should be kept well open with a purgative, preferably a saline. It may be necessary to assist their action with enemata, and if there is retention of urine the bladder should be relieved either by means of the catheter, or by compression through the abdominal wall.

Tumours of the Spinal Cord

Tumours of the spinal cord are comparatively rare. Occasionally sarcomata have been met with in the horse, ox and dog, and gliomata and lipomata in cattle; cholesteatomata, chondromata and melanomata have also been met with in the horse, the last-named usually in greys.

Symptoms.—These tumours are usually slowly developed and cause gradual interference with the spinal functions. In the early stages there may be some increased pain posterior to the growth, but this is eventually followed by reduced sensation and weakness of the hind limbs. Animals lie down a lot and rise with difficulty, and dogs are unable to jump. If they are allowed to jump off the table or chair their hind limbs collapse under them. Paraplegia usually follows with interference with the functions of defæcation and micturition.

Similar symptoms may also be produced by compression of the spinal cord as the result of tuberculosis, actinomycosis, parasites, and abscesses. The latter may be an extension from poll-evil, fistulous withers, or other injuries to the back.

Myelitis Spinalis; Inflammation of the Spinal Cord

Inflammation of the spinal cord is not a very common condition. It occurs most frequently in the dog and next in frequency in the horse. In the majority of cases it occurs as a complication of some bacterial affection such as distemper, rabies, and influenza. In some cases it may be tubercular in origin. A similar condition with degeneration of the spinal cord occurs in the advanced stages of trypanosome affections. Myelitis may also occur as the result of mechanical injuries—this form has been already dealt with in the preceding article on "Spinal Concussion," and will not be further considered here. A peculiar form of inflammation of the spinal cord occurs in dogs, sometimes as the result of obstinate constipation and impaction of the colon, possibly as the result of an auto-intoxication.

Symptoms.—Symptoms vary according to the extent of the lesions, which may involve either the entire thickness of the spinal cord or only one portion of it. The former is the more common form. The symptoms usually commence with some debility, particularly of the hind limbs. Affected animals lie down for longer periods than usual and have some difficulty in rising. They tire very rapidly when put to exercise or exertion. Over the areas posterior to the affected portion of the cord there is diminished sensation, which gradually becomes more pronounced until there is complete loss of sensation, and finally paralysis of the hind limbs occurs (paraplegia). The larger animals are quite unable to rise, and small animals, in attempting to move about, trail their hind limbs along the ground. At the same time there is relaxation of the sphincters and involuntary passage of urine and fæces, while occasionally retention occurs.

The condition may extend forward. Then the fore limbs become gradually affected in a similar way until paralysis of all the legs is set up. Before paralysis of any area is complete there may be twitchings and spasms of the various muscles, and this is followed by a soft flaccid condition of the muscles and complete loss of the reflexes.

In those cases in which a portion only of the cord is involved (*myelitis transversalis*) there may be only one limb affected, but the condition usually extends and soon involves other areas. There is usually no alteration of temperature, and the appetite is often fairly well maintained in the early stages.

Course.—In acute cases the course of the disease is fairly rapid, and it may prove fatal in from several days to a week. Other cases run a more protracted course and may last from six to eight weeks. In these prolonged cases various complications are likely to arise, such as pneumonia, enteritis, cystitis. Recovery is not very hopeful, particularly in the larger animals. In the dog, however, recoveries are more frequent after a course which may extend to three months. Herein, however, lies the difficulty of prognosis in that some dogs will appear to be making good progress and be subject to a relapse after a couple of months, the case then making rapid progress for the worse and terminating fatally. In those cases, however, due to impaction of the colon a gradual recovery takes place in about six to eight weeks under appropriate treatment.

Treatment.—The patient should first be placed under comfortable conditions and provided with a good bedding, but must not be allowed to remain in one position too long. He should be turned over every two or three hours to prevent bed-sores, and the limbs should be massaged. If there is difficulty in the passage of fæces, the latter should be removed manually in the case of the larger animals, and by enemata sometimes assisted with small parturition forceps in the smaller animals. The bladder must be relieved either by means of the catheter, or by pressure on the bladder wall per rectum in the larger animals or through the abdominal wall in the smaller animals. If there is involuntary passage of fæces and urine, thorough cleanliness must be observed, and the soiled skin be dusted with kaolin or fuller's earth in order to prevent blistering. Medicinally a laxative should first be given, subsequently iodide of potassium and nux vomica should be given twice daily. After two or three weeks' administration, this may with advantage be replaced for an interval of one week with liquor arsenicalis (Fowler's Solution), or with liquor arsen. et hydrarg. iodid. (Donovan's Solution).

External applications of counter-irritants may also be of service, such as the application along the middle of the back of a stimulating liniment or a mild blister. At other times the application of the electric battery may be resorted to. In those cases following impaction of the colon in the dog, the writer usually obtains excellent results by the administration of a purgative dose of calomel (gr. i.-iv.), followed by a course of spts. ammon. arom. mm. xv.-ʒi. and tinct. nucis vom. mm. v.-x., administered twice daily, alternated fortnightly with Fowler's Solution, mm. v.-x., twice a day, and accompanied by the application to the lumbar region of a blister of biniodide of mercury ointment, 1-8.

Parasites of the Brain and Spinal Cord

One parasite is frequently met with in the brain, viz. *Cœnurus cerebralis*, in sheep, goats and cattle, and more rarely horses may be affected.

The following parasites are also occasionally met with :

Echinococcus cysts, in horses and cattle.

Cysticercus cellulosæ, in the pig, and rarely the dog.

Cysticercus bovis, in calves.

Cysticercus pisiformis, in dogs.

Immature *Strongylus vulgaris*, in horses.

Larvæ of *Gastrophilus*, in horses.

Larvæ of *Hypoderma bovis*, in the ox and horse.

Larvæ of *Œstrus ovis*, in sheep.

Cerebral Cœnurosis (Gid, Sturdy, Turn-sick).—Gid is an affection frequently met with in sheep, and brought about by presence in the brain, or more rarely in the spinal canal, of a bladder-worm, the *Cœnurus cerebralis*, which is the cystic stage of the *Tœnia cœnurus*, a tapeworm of the dog (*q.v.*) The parasite may also be met with in other animals than the sheep, viz. the ox and goat, and more rarely the horse.

Infection takes place by the ingestion with food or water of eggs which have been liberated by the rupture of the mature segment of the tapeworm voided by a dog. The egg hatches out in the stomach, and the embryo, by means not well understood, eventually finds its way to its predilection seat in the brain, or occasionally in the spinal cord. There are several views as to the manner in which the parasite arrives at its destination. One view is that it migrates by way of the connective tissues. Another view is that it penetrates into the blood-vessels, and is carried to the brain by the blood-stream. On arrival in the brain the embryos develop into cysts varying in size from that of a pea to the size of an egg in sheep, and they may be single or there may be several cysts. As many as thirty have been recorded in a case by Huzard. In cattle the cysts sometimes attain much larger dimensions, up to the size of a closed fist, and they are frequently multiple. In the horse they are usually single. The common seat of the cysts is on the upper surface of the cerebrum or between the cerebral hemispheres, while occasionally they are met with in the lateral ventricles, and at other times in the cerebellum. As they increase in size they cause pressure on the brain substance, which may become atrophied and cause loss of brain functions. When in the roof of the cranium pressure may also be exerted on the cranial bones, which in sheep may become quite thin and soft, and yield to pressure with the thumb.

In rare cases the parasites are met with in the spinal canal, causing derangement of spinal functions.

Symptoms of Gid in Sheep.—Symptoms may be set up in the early stages of invasion of the parasite as the result of local inflammation when the parasite is arriving at its resting-place. The symptoms set up are those of meningitis and encephalitis. The affected sheep isolate themselves and become affected with loss of voluntary control, often turning in circles with the head twisted to one side. At other times they make erratic movements forward, stumbling and falling, and in some instances are unable to rise. In those cases it is usually fatal in about a week after the onset of symptoms, or about three weeks after infection. These early symptoms, however, are frequently much less pronounced, and in the majority of cases pass unnoticed. At a later stage symptoms are again set up owing to disturbance of functions, brought about by the increase in size of the parasites. This may be in from four to six months after infection. At this stage affected sheep usually isolate themselves, are unsteady in gait, and have a vacant expression. The head may be turned to one side, or elevated, or pressed down between the fore limbs, while some sheep will stand with their head pressed against an obstacle, apparently unable to leave it. On attempting to move, the sheep usually turns round in circles continuously to one side, the circles getting smaller as the condition gets worse. It then becomes giddy and may be unable to stand. At other times high-stepping is observed (trotters), while at other times somersaults are turned forwards or even backwards ; spasmodic leaping movements are sometimes met with (high-flyers). Pivoting on one fore limb may occur, and less frequently rolling on the long axis of the body.

During an attack of gid the appetite is variable. Some affected sheep will eat fairly consistently with intermittent epileptiform seizures, with frothing at the mouth and dribbling of saliva. There is great loss of condition. On manipulating the head one may frequently discover areas of the bone which yield to pressure. This pressure may bring about epileptiform convulsions or collapse ; at other times it apparently causes no inconvenience. This softening usually, but not invariably, marks the location of the cyst, and in the case of animals turning in circles, will generally be found on the side corresponding with the inside of the circle. At the same time one frequently notices paralysis of muscles or limbs of the opposite side of the body. The animal thus turns towards the side affected with the cyst. The cyst is usually in the frontal lobes in those cases where the head is depressed and pushed forwards. In animals performing backward somersaults the cyst will usually be found between the cerebrum and the cerebellum.

When the parasites are in the spinal cord symptoms are due to spinal compression, and consist mainly of paralysis of the hind limbs, with capricious appetite and considerable loss of condition.

In cattle, symptoms are set up similar to those in sheep, with interference with locomotion, circus movements occasionally following, and rolling gait. The head is usually depressed to one side. Occasionally softening of the bone may be met with as in sheep. There is progressive wasting and loss of appetite.

In the horse, symptoms may resemble those of meningo-encephalitis with irregular movements and emaciation, as in tumours of the brain.

In all cases allowed to run the natural course death takes place as the result of exhaustion and anæmia.

Treatment.—Curative measures are not very satisfactory, and in the case of food animals it is advisable to slaughter them as soon as the condition is noticed if they are in good bodily condition. In the chronic stage surgical interference and removal of the cyst by trephining may be attempted, but it is only likely to be successful in those cases where the cyst can be localized and is somewhat superficial, and where the animal still remains fairly strong and is feeding reasonably well. The writer has operated in quite a considerable number of cases in sheep, with success in from 50 to 60 per cent of cases. For details of surgical treatment the reader is referred to the surgical section, p. 794.

Prevention.—In districts where the disease is known to exist, preventive measures should be directed towards the dogs belonging to the farmer himself and his neighbours when obtainable. They should be dosed with tæniafuges, and should be kept in close confinement until the worms have been expelled. The fæces and parasites should then be destroyed by burning. This process should be repeated periodically several times a year. Care should also be taken that dogs should not be allowed to eat the heads of sheep or other animals that have been affected with the condition.

G. H. W.

Diseases of the Peripheral Nerves

Neuritis; Inflammation of the Nerves

This condition is fairly common in domesticated animals and may affect either the nerve sheath or the axis cylinder (the true nerve substance). When the inflammation commences with the sheath of the nerve and the surrounding connective tissue it is called **perineuritis.** If it commences with the true nerve substance, it is called **interstitial neuritis.** When the affection is a degeneration of the nerve it is called **parenchymatous neuritis.**

Neuritis may be brought about by varied causes, the commonest one being bacterial infection. It may also be brought about by poisons such as bacterial toxins. An example of this is furnished by the part played in the causation of roaring in horses as the result of strangles and other catarrhal affections. Chemical poisons may set up neuritis as exemplified by lead (neuritis of extensors), arsenic (optic neuritis), and mercury. Inflammation of nerves may occur as the result of extension of inflammatory phenomena from surrounding tissues. It may also be due to mechanical causes, as by direct injuries from wounds or bruises or fracture of contiguous bones (radial paralysis). Compression of nerves may cause the condition as in the case of injuries of surrounding tissues or development of tumours or fibrous tissue (amputational fibroma after neurectomy). The stretching of nerves has a similar effect, as in radial and obturator paralysis due to stretching by the callus resulting from fracture of a rib and the os pubis respectively. Nervous exhaustion may also bring about neuritis. An example of this is furnished in one form of suprascapular paralysis in young agricultural horses working in the plough field. The horse working in the furrow frequently walks with one limb on top of the ridge, and the continued effort to raise that limb produces exhaustion of the suprascapular nerve, with neuritis followed by paralysis. Exposure to cold also starts up neuritis in certain subjects. The exact manner by which cold operates is unknown, but the condition is probably rheumatic in character (lumbago, sciatica).

Effects of Neuritis.—These vary considerably with the character of the nerve affected. In the case of a sensory nerve there is acute pain produced over the area supplied by that nerve and may be followed by anæsthesia. In the case of a motor nerve at the earlier stages there may be spasms of the muscles supplied by the nerve, and as the condition becomes more severe it may result in paralysis. In the case of a mixed nerve, local hyperæsthesia and muscular convulsions may be followed by anæsthesia and paralysis.

The effects of lesions of the nervous system on the peripheral nerves depend upon the character and position of the lesions, *i.e.* whether the lesion irritates or stimulates the nerves on the one hand, or whether it destroys the function of the nerves. As to position, the effects will vary according to whether the lesion involves the brain, the spinal cord, the trunk of the nerve, or the nerve terminals. Thus if a motor nerve or its centre is stimulated or irritated, muscular convulsions or spasms are produced, while irritation of a sensory nerve causes pain or increased sensibility of the part supplied by the nerve. If the lesion destroys the nerve or its centre, then motion or sensation is lost.

The following *symptoms* may therefore be variously produced :

Spasm—abnormal violent muscular contractions as in cramp. Spasms are called **tonic** or **tetanic** when they are violent and continuous, and **clonic** when less violent and more or less rapidly intermittent.

Tremors or tremblings—very small involuntary, intermittent contractions as in rigors.

Paresis—a degree of defective innervation where motion is impaired but not lost,

Paralysis—the loss of voluntary or involuntary muscular movement through defective innervation. (Sometimes **muscular paralysis** is spoken of when the defect is muscular and not nervous in origin.)

Hemiplegia—loss of voluntary motion of one side of the body due to lesions of the opposite side of the brain or the opposite half of the spinal cord. **Cross hemiplegia** is paralysis of the muscles of one side of the head and of the opposite side of the body, and is cerebral in origin.

Paraplegia—loss of voluntary movement of a transverse half of the body, usually due to spinal lesions.

Monoplegia—a very limited paralysis, as of a limb, one muscle, or of one side of the face.

Hyperæsthesia—**neurasthenia**—increased excitability or sensitiveness of any part of the sensory nervous system, and may be referred to the periphery. In this way pain may actually be referred to an amputated limb.

Hemi-æsthesia—increased nervous irritability and sensitiveness of one side of the body.

Paræsthesia—increased sensation of a transverse half of the body.

Neuralgia—pain along the course of a nerve.

Anæsthesia—absence of sensation; may be local or general, partial or complete.

Hemi-anæsthesia and **Paranæsthesia** — loss of sensation of one side or a transverse half of the body respectively.

Muscular Atrophy—loss of volume of muscular tissues. This may be due either to lesions of trophic nerves causing interference with nutrition of the muscles (*trophic neurosis*) or to motor paralysis (atrophy of inaction).

Symptoms produced as the result of lesions in the brain are called **encephalic symptoms.** When due to spinal lesions they are called **spinal symptoms.** It is not always easy to distinguish between them, but the following points may be of material assistance for differential diagnosis :

Encephalic Origin.	*Spinal Origin.*
Hemiplegic grouping of symptoms.	Paraplegic grouping of symptoms.
Area supplied by cranial nerves.	Area supplied by spinal nerves.
"Head" symptoms frequent, such as frenzy or coma.	"Head" symptoms rare.
Muscle atrophy slight or absent.	Muscle atrophy marked.
Little tendency to form bed-sores.	Bed-sores and sloughs common.

Most forms of paralysis in animals are brought about by injuries or other traumatic causes, consequently they are surgical conditions and will be considered in greater detail in the surgical section dealing with the special areas involved, consequently only brief reference will be made to them here.

Optic Neuritis and Optic Paralysis.—These conditions are most frequently due to some disease of the brain or the optic centres. It may also occur in the course of some specific affections and in lead poisoning. It usually results in amaurosis and blindness, often with no noticeable structural change of the eyeball. At other times hyperæmia of the retina may be observed. The writer has frequently observed the condition in Jersey cattle due to unknown causes (p. 771).

Paralysis of the Motor Nerves of the Eyeball (2nd, 4th, and 5th Pairs of Cranial Nerves).—This condition is also generally due to some traumatic cause affecting the head or brain, or the presence of tumours or inflammatory processes. It results in drooping eyelids, dilated pupil, and various alterations in movement of the eye, such as squints, etc.

Paralysis of the 5th Cranial Nerve.—This condition is generally due to some affection of the root of the nerve as may be brought about by concussion or tumours in the brain, or in the case of the dog in rabies and distemper. In these cases it is generally bilateral. It may also be due to injuries to one of the branches of the nerve, in which case it is generally unilateral. The symptoms produced depend upon the branch of the nerves affected, and may consist of an increase of sensibility of the face, which may be followed by anæsthesia and also interference with mastication. Infection through the dental nerves may set up neuritis of the affected tooth, and extending sympathetically along the whole side of the face, cause relaxation of the muscles of one or both sides of the face. The writer has seen this condition in the horse due to diseased upper molar tooth, in which the upper lip of the diseased side was drawn somewhat to the opposite side owing to loss of muscular tone.

He has also seen a similar condition of neuralgia in a dog following pyorrhœa and the extraction of an upper molar tooth. In this case both sides of the face were affected, the upper lips were slightly pendulous. There was evidently continuous pain, as the result of which the dog constantly rubbed the lips along the ground or against obstacles until he actually injured them. There was simultaneously a slight drooping of both upper eyelids. Paralysis of the inferior maxillary division of the 5th nerve produces paralysis of the muscles of mastication, which also undergo atrophy. This may be unilateral or bilateral.

Paralysis of the Facial Nerves (the 7th Cranial Nerve).—This condition is generally brought about as the result of injury in the region of the posterior border of the jaw. It causes paralysis of the muscles of the side of the face (facial paralysis). The condition is dealt with in detail on p. 810.

Paralysis of the Auditory Nerve.—This condition may be congenital, resulting in permanent deafness, as is frequently the case in albinos of various species. It is a very common condition in white cats, some of which may have a pigmented iris. Inflammation of the 7th nerve resulting in paralysis and deafness may also follow inflammation of the middle and internal ear, and is then generally associated with twisting of the head and vertigo (Menière's Disease).

Paralysis of the Vagus, Pneumogastric or the 10th Cranial Nerve.—This is not a common condition, but may occur in the course of bacterial affections, or may be due to lead poisoning or lathyrism. The principal symptoms of this condition are associated with the pharynx and œsophagus, which receive their motor nerves from the vagus. There is difficulty in swallowing, and often choking, with the return of the food by the mouth or nostrils, also partial paralysis of the larynx resulting in stertorous breathing. This is frequently followed by gangrenous pneumonia, owing to the passage of food and saliva down the trachea. Treatment is not at all satisfactory. A course of nerve tonics such as strychnine and arsenic may be administered, or in the case of lead poisoning a course of iodide of potassium.

Paralysis of the Recurrent Nerve.—This is the condition that brings about roaring in horses, owing to paralysis of the left side of the larynx; it has been considered in detail on p. 378.

Paralysis of the Suprascapular Nerve.

Paralysis of the Radial Nerve.

Paralysis of the Sciatic Nerve.

Paralysis of the Obturator Nerve.

Paralysis of the Tail.

For the above-named conditions see Surgical Section, Vol. II. pp. 729-733. G. H. W.

DISEASES OF THE MUSCULAR SYSTEM

Diseases of the muscles other than muscular rheumatism and parasitic diseases are generally regarded as surgical conditions, and are dealt with in the surgical section of this work, consequently only passing reference is made to them here.

Myositis (Inflammation of Muscle)

Several forms of inflammation of muscle are recognized, viz. acute myositis, chronic myositis, and rheumatoid myositis or muscular rheumatism.

Acute myositis may occur as a primary affection, generally as the result of injury or rupture of a muscle. It may also be due to extension of inflammation from diseased bones, or it may be secondary to infectious diseases such as pyæmia and occasionally tuberculosis, and to animal parasites, as in trichinosis. The common symptoms consist of pain, tenderness, and swelling at the seat of the inflammation. It may lead up to exudation and even abscess formation.

Acute Myositis of the Masticatory Muscles in the Dog.—The above rare peculiar condition, due to unascertained causes, has been met with by the writer in a number of dogs. The condition is acute in its onset and is associated with great pain in the region of the head and complete closure of the mouth (trismus). The affected dog cries out when a hand is placed on his head, and any attempt to open the mouth forcibly fails and causes pain. The temporal muscles are hot and considerably swollen. The patient is unable to take either solids or liquids, and has a very anxious expression of countenance. The temperature is raised to 105° F. The painful condition lasts two or three weeks, and no other muscles than those of the head and face appear to be involved. The patient is usually anxious for food, and if artificially fed with liquids through a pouch of the lip will take food quite readily. After several weeks the pain subsides and the swollen muscles become smaller and gradually undergo atrophy, so that ultimately there is a deep depression on each side of the head and the cheeks also become thin. By this time the dog will usually learn to drink by suction if liquids are placed in a deep vessel. He will also attempt to press solid food into his mouth between the teeth. After two or three months the teeth can be slightly separated and the trismus becomes gradually relieved, so that by the end of six months the dog can feed normally and can actually masticate bones.

Treatment.—In the early stages the pain and fever must be controlled by aspirin or other salicylate in combination with tincture of nux vomica. Any constipation should be relieved by small doses of castor oil or paraffinum liquidum. Locally, relief is afforded by hot fomentations and the application of the combination of lin. belladonnæ and lin. saponis āā. When the painful stage is passed the administration of tincture of nux vomica and liquor arsenicalis may be resorted to. The use of electricity in some cases by the writer did not appear to produce any better results than were obtained in other cases without it. Throughout the period when the dog is unable to feed itself artificial feeding must be resorted to, but, as already stated, dogs with this condition will soon learn to drink if they are provided with their liquids in a deep basin instead of the usual shallow dish. A diet of milk or eggs and milk suffices until solid food can be voluntarily taken.

Chronic Myositis.—This condition is usually associated with inflammation of fibrous tissues, to which the term *Fibrositis* is applied. The causes of the condition are varied and often obscure, but they are frequently of rheumatoid origin. It may give rise to distortion of various parts of the body, such as the neck or limbs. It may also be brought about in horses by azoturia, and result in formation of breaches of the muscle in various parts of the body, such as the quarters, croup or back, or the shoulder.

Treatment is not very hopeful. It consists in the administration of a purgative and a course of salicylates and iodides, and the external application of counter-irritants.

Muscular Rheumatism.—See "Rheumatism," p. 248.

Muscular Atrophy.—Simple atrophy of muscle fibres may be due to defective nutrition—starvation—and occur in the course of wasting diseases such as tuberculosis, glanders, nephritis, diabetes, etc., and in senility. It may also occur as the result of paralysis, particularly of the trophic nerves. In the latter case the atrophy is local, involving a single muscle or group of muscles. Atrophy of muscles of the limbs may also result from inaction due to lameness arising from affections of other structures, *i.e.* shoulder atrophy in foot lameness in horses.

In muscular atrophy the muscles become reduced in volume, pale, and flabby, and unable to perform their functions to the normal degree, thus causing general or local muscular debility and wasting.

Treatment of muscular atrophy depends upon the causes. When due to systemic affections, the latter must be treated and a generous supply of nutritives prescribed generally, including cod liver oil and iron salts. When due to local causes, the intramuscular injection of liq. strychninæ hydrochlor. may be tried, or the application of counter-irritants, such as blisters, setons, or the actual cautery. Electricity is sometimes helpful.

Fatty Degeneration of Muscle.—In this condition fat is produced within the muscle fibres, probably at the expense of the protoplasm. It most often occurs in heart muscle, but it may also be met with in voluntary muscles. In the latter situations it may be seen in very young animals of any species, particularly in pigs. It may also occur in adult cattle, affecting large areas of the voluntary muscle. The causes of the condition are unknown, but in some cases it is accompanied by anæmia; at other times it is associated with excessively high feeding. In cattle fatty degeneration of muscular tissue may be found in the abattoir without its previous existence having been suspected, large tracts of muscle having been converted almost entirely into lumps of fat.

Granular or Parenchymatous Degeneration of Muscle.—This condition may arise in the course of fevers and other acute conditions. The most common example is met with in azoturia of the horse. It may also occur as the result of poisoning by such agents as phosphorus, and may lead to fatty degeneration.

Parasitic Affections of the Muscular and Connective Tissues

Sarcosporidia; Muscular Sporozoa

Three species of sarcosporidia or psorosperms have been described affecting domesticated animals, viz. *Sarcocystis miescheri* and *S. tenella*, which affect the muscular tissues of the pig and sheep respectively, and *Balbiania gigantea*, which affects the connective tissues in sheep, ox, goat, and pig. Some authorities consider that there is not sufficient ground for separating sarcocysts into these classes, and regard them as the same parasites simply growing in different tissues.

Sarcosporidia in the Pig.—The muscles of the pig may become invaded with the *Sarcocystis miescheri*, producing what have been described as Miescher's tubes or Rainey's corpuscles. They rarely cause any symptoms during life, but are sometimes responsible for debility or paresis of the hind limbs, together with a skin rash and some fever. Infection probably occurs by the ingestion of infected water or food material.

The sarcocysts are most frequently met with in course of meat inspection, and since it is possible the human being may be infected it assumes greater importance. Pork which is badly affected becomes darker in colour and has a somewhat granular appearance, being dotted with small elongated bodies up to $\frac{1}{12}$th of an inch in length. Under the microscope these structures are seen to be within the muscle fibre and to consist of a thin capsule containing numerous smaller bodies either kidney-shaped or circular. In some cases calcification of the capsules takes place.

Sarcosporidia in the Sheep.—The sarcocyst of the sheep has been called the *S. tenella*, and is very similar to that occurring in the pig. The cysts are, however, somewhat smaller, and are rarely visible to the naked eye. They are most frequently found in the muscles of the inner side of the thigh and in the cardiac muscles, but they may be found in the muscle tissue anywhere.

They may give rise to symptoms of anæmia, emaciation, general unthriftiness, and sometimes difficult respiration; but in many cases no symptoms whatever are exhibited.

Sarcosporidia have also been observed in the horse, ox, and the dog.

In the **horse** they have been associated with urticaria-like swellings on the skin and sometimes induration of the tongue, thus interfering with feeding and causing general loss of condition.

In **cattle** similar symptoms may be set up.

The sarcocyst of the *connective tissue* is known as *Balbiania gigantea*, and affects sheep especially. It has also been met with in the ox, pig, and goat. The cyst occurs in the form of a small nodule varying in size from a barley grain up to the size of a hazel nut, and contains the characteristic crescentic corpuscles. The common situation for these balbianiæ is the œsophageal wall, and after that they are met with in the tongue, pharynx, cheek, neck, abdomen, and thighs, occasionally under the pleura and peritoneum.

This parasite is said to cause difficulty in feeding and consequently to set up emaciation. At other times it causes dyspnœa, from œdema of the glottis. It occasionally occurs in the form of an epizoötic.

Treatment of sarcosporidiosis is not very hopeful, but potassium iodide may be tried.

Muscular Cysticercosis; Measles

The term "measles" in the lower animals is applied to infestation of the muscular tissue with various parasitic cysts. In the ox the parasite responsible is the *Cysticercus bovis*; in the sheep it is due to *Cysticercus ovis*; while in swine, measles is due to *Cysticercus cellulosæ*. The latter parasite has also been observed in the dog and cat.

Measles of the Ox.—*Cysticercus bovis*, the cause of measles in cattle, is the cystic stage of the *Tænia saginata* (*T mediocanellata*), which is the unarmed tapeworm of man.

Appearance of the Parasite.—The vesicle or cyst is elliptical in shape and attains a length of about ¼ of an inch with half that width. It is found embedded in the connective tissue of the muscles. The bladders are greyish-white in colour with a transparent wall, and they are surrounded by a fine connective-tissue capsule. Through the transparent wall can be seen an opaque spot, which is the invaginated head or scolex of the future tapeworm. When the head is everted and examined under magnification it is found to possess four large suckers and a central depression, but no hooklets.

Infection takes place as the result of cattle taking up eggs or segments of the tapeworm voided by a human being, the host of *T. saginata*. They may be taken in with contaminated food or water. The eggs may remain infective on damp ground for a very long time, and since one segment or proglottis is stated to contain as many as 30,000 eggs it will thus be seen that one infested man may very seriously infect pastures in those localities where human excreta are improperly disposed of. When the eggs are ingested the shells are dissolved by gastric juice and small embryos are hatched out. These bore through the stomach wall and are distributed over the body by the blood stream. They become arrested usually in the muscular tissue and there develop into cysts or hydatids. The development of the cyst is very slow, and in two months it is only the size of a pin's head, and continues to increase in size for five or six months. Old cysts in the muscle tissue may die and undergo degeneration and become calcified, giving rise to the condition which is called "Dry Measles." The common seats of the parasites are: (1) the internal pterygoid muscles; (2) the heart; (3) the tongue; (4) the whole voluntary muscle in bad cases. In rare cases they may be found elsewhere, as in the liver, spleen, kidney, and the anterior chamber of the eye.

Symptoms.—As a rule there are no symptoms set up during life, and they are usually unsuspected till they are discovered in the abattoir.

The chief importance of the parasite, therefore, lies in the fact that a human being eating infested beef raw or improperly cooked would become the subject of tapeworm. Cooking, however, readily destroys the parasite, a temperature of 50° C. being sufficient to kill it.

Measles in beef is very rarely met with in the British Isles, but is more common on the Continent of Europe, particularly in Germany, and also in India.

Treatment.—No curative measures are possible even if the presence of the parasite is diagnosed during life. Preventive measures, however, may be taken, and consist principally in the proper disposal of human excreta. If, as in country districts, it is intended to be applied to land as manure it should first be mixed with quicklime. Any person affected with tapeworm should be suitably treated.

Measles in Swine.—*Cysticercus cellulosæ*, the cause of pig measles, is the cystic stage of the *Tænia solium*, the armed tapeworm of man. The cyst may also be met with in the dog and cat, occasionally in deer, and rarely in the human being. In the latter case it is usually considered to be the result of auto-infection.

Appearance of the Parasite.—The vesicle is found in the muscles, and closely resembles *Cysticercus bovis*. It is about the same size, but its wall is rather more transparent. It possesses a similar opaque spot with a minute hole, which is the invaginated head or scolex. On squeezing out the head and examining under magnification, it is seen to possess four suckers and a double crown of hooklets.

Infection with this cyst takes place as the result of swine ingesting food or water contaminated with eggs or segments of *T. solium* voided by an affected human being. The liberated embryos invade the tissues by way of the blood stream, and in about three weeks attain the size of a pin's head. They become fully developed in from two to three months. Old cysts may die and undergo calcification. The predilection seats of the parasite are: (1) the abdominal muscles; (2) the muscular part of the diaphragm; (3) the tongue; (4) the heart; (5) the masticatory muscles; (6) the intercostal muscles; (7) the muscles of the neck; (8) all the muscles in bad cases. On rare occasions cysts may be found in the eye or brain or internal organs. In severe cases the parasites may be very numerous, much more so than in measles of the ox.

Symptoms.—In mild cases no symptoms are to be discovered during life. In bad cases, however, various symptoms may be set up. Affected animals become debilitated, lose their appetite, and appear to be generally unthrifty. Owing to the frequency with which the tongue becomes infected, that organ should be examined, particularly along the edges and the under surface and the *frænum linguæ*, where cysts may be felt with the fingers under the mucous membrane about the size of a small pea. A cyst may also be observed in the anterior chamber of the eye or even under the conjunctiva.

The chief importance of this parasite lies in the fact that a human being eating infected pork uncooked or insufficiently cooked would become infected with *T. solium* in three to four months. Thorough cooking, however, with a temperature of 50°-55° C. extending to the centre of the joint is sufficient to destroy it.

Treatment.—*Curative* treatment is of no avail. *Preventive* measures are the same as recommended for *Cysticercus bovis*. Although cooking destroys the parasite it is unwise to pass affected carcases for human food, since infection may take place as the result of handling infected flesh. An efficient system of meat inspection is therefore necessary.

Measles in Sheep.—*Cysticercus bovis*, which is the cause of measles in sheep, is the bladder-worm stage of *Tænia ovis*, an armed tapeworm of the dog. It is a very rare parasite, but is most frequently met with in America. The cyst somewhat resembles *Cysticercus cellulosæ*, but is rather smaller, rarely exceeding one-fifth of an inch in length. The common seats of the parasite are the heart, the diaphragm, the tongue, and muscles of mastication. In bad cases, however, it may be found in the muscle tissue anywhere. Infection is due to the ingestion of food or water contaminated with the eggs or segments of *T. ovis* voided by an infected dog.

No symptoms have been observed during life, and the parasite has only been observed in the abattoir. Since the human being is not liable to infection with *T. ovis*, measles in sheep is of less importance than measles in the ox or pig.

Preventive measures in districts where the disease exists consist in the administration of tæniafuges to the dogs, which should be kept under control while the drug is in operation, in order that the expelled worms may be collected and cremated.

Nematodes of the Muscular and Connective Tissue

Trichinella spiralis; Trichina spiralis.—*Trichinella spiralis* is a small nematode worm, the adult stage of which is about one-eighth of an inch long, and is met with in the intestines of the pig and other mammals (rats, mice, cats, and dogs). Infestation takes place as the result of the ingestion of flesh containing larvæ of *T. spiralis*. From the larvæ the adult worms are developed in several days. Fertilization then occurs, and within a week living embryos are deposited between the intestinal villi or in the glands of Lieberkühn. This process continues for five or six weeks, during which time as many as ten thousand larvæ may be produced by one female. The larvæ then penetrate into the lymphatics and are carried to the blood stream, by which they are distributed throughout the body. They come to rest in the voluntary muscles, which may become attacked in ten or twelve days after the ingestion of infected meat. The muscles chiefly affected are, in the order of frequency:

(1) The muscular part of the diaphragm.
(2) The muscles of the tongue and larynx.
(3) The intercostal muscles.
(4) The muscles of the neck and shoulder.
(5) The abdominal muscles.

Tendons are not affected, nor is heart muscle.

The lesions in the muscle consist of the encysted and coiled larvæ, about one-third as long as the adult. The larvæ invade the muscle fibre, which they distend, and a lemon-shaped capsule develops around it. In this situation the young worms are invisible to the naked eye, and microscopic examination is necessary for their detection. If, however, the host lives a sufficient time the capsules become calcified, and may then be visible to the naked eye. When flesh containing living muscle trichinæ is eaten in the raw or imperfectly-cooked condition by a human being or a pig, the muscle trichinæ are liberated in the bowel, becoming mature in two or three days. Within a week the females begin to produce young, and so continue the life-history.

Symptoms.—Symptoms of bowel infection in swine are only set up when the parasites are present in considerable numbers, and consist principally of general depression and fever, and sometimes diarrhœa. It may occasionally prove fatal.

The second stage of symptoms occurs about ten days after infestation and lasts about a fortnight. In this latter period parasites are migrating throughout the body, and set up the condition known as *trichinosis* or *trichiniasis.* During this stage in badly-infested animals general stiffness and muscular pains are set up, with interference with mastication and swallowing; often the affected pig lies with his limbs outstretched. When the parasites have become encapsuled the symptoms subside, and affected swine may then become thrifty and put on flesh. Pigs are usually infected as the result of eating trichinous rats or trichinous pig's flesh.

Diagnosis of trichinosis in living swine is rarely made. The symptoms described are those observed in experimental infection.

Treatment.—No *curative* measures are of any service. *Prevention* consists in destroying all rats which may be intermittent carriers of the parasites, and boiling all butcher's offals fed to swine. All pork found to be trichinous should be destroyed. Cooking with a temperature of 55° C. or over destroys the vitality of the parasite, while boiling, allowing fifteen minutes for each 1 lb. of the joint, will usually sterilize it. An efficient system of meat inspection is essential for the protection of human beings.

Ollulanus tricuspis.

Onchocerca reticulata—Spiroptera reticulata—Filaria reticulata. — *O. reticulata* is a very fine thread-worm up to 2 feet in length, which may be met with in horses, coiled round in muscular, tendinous, and ligamentous tissues. It occurs on the Continent of Europe and in Persia. The method of infection is unknown.

Symptoms.—This worm sets up fibrous tumours under the skin and between the tendons, generally fusiform in shape and from 1 to 2 inches long. Old lesions may undergo calcification. Its common situation is in the flexor tendons of the fore limbs, the suspensory ligament, and in the wall of the metacarpal artery. In some cases the lesions produce lameness, either as the result of interference with the action of the tendon or ligament, or by pressure upon the nerves.

The *treatment* is excision.

Onchocerca cervicalis.—This parasite is also found in the horse, and sets up similar lesions in the ligaments of the neck (*ligamentum nuchæ*). It apparently causes no symptoms during life.

Onchocerca gibsoni—O. lievalis. — *O. gibsoni* infests cattle and produces in beef the condition known as white kernels or worm nests. The parasite is a long, thin worm somewhat similar to *O. reticulata* of the horse. The length of the female is estimated at 3 feet, but a complete worm has never been obtained, owing to the intricate convolutions through dense connective tissue of the nodule. The male is about 2 inches long. The adult worms are found in firm, nodular tumours from ½ to 2 inches in diameter in the superficial and intermuscular connective tissue, most frequently in the brisket, in the flank, and around the stifle-joint. The method of infection is not definitely known. It is probable that the embryos are shed into the blood stream, through which they are sucked up by biting flies, which transmit them to other animals.

Symptoms.—The parasites, even in a heavy infection, appear to give rise to no ill effects, and the general health of affected cattle is apparently unaltered. The existence of the parasite is of principal importance from the point of view of meat inspection.

It is met with principally in Australia, America, and in Northern Africa.

Onchocerca gutturosa.—This parasite also affects cattle, and is met with affecting the aorta of cattle in India. It is possible that it is the same parasite as *O. gibsoni.*

Onchocerca capræ.—This parasite has been described as affecting the tongue in goats, setting up similar lesions.

Species of **Filaria** infect the connective tissue in dogs, but do not cause any symptoms.

They include *Filaria acutiuscula* in Italy and Brazil, and *Filaria recondita* (*F. lewisi, Hæmatozoon lewisi*) in Italy, Malta, and India.

G. H. W.

DISEASES OF THE SKIN

Cutaneous diseases may be defined as "aberrations of the skin as indicated by alterations in its appearance, functions, sensibility, and texture." They may be classified into diseases of (1) the circulation, (2) nutrition, and (3) sensibility. If classified according to the structure of the skin they may be divided into (1) diseases of the epidermis, (2) diseases of the rete mucosum, (3) diseases of the glandular apparatus, and (4) diseases of the hair follicles and hair.

The *general symptoms* of skin diseases are principally indicated by (*a*) Changes in the colour, which may be due to alterations of the circulation, causing pallor or redness from hyperæmia as seen in erythema, urticaria, and eczema. These alterations of colour, however, are not readily observed in animals with a pigmented skin. Changes of colour may also be due to alterations of the pigmentation of the skin, the best example of which may be met with in the black pigmentation often seen in follicular mange in the dog and the pig, and also melanosis in grey horses. (*b*) Changes of texture. These may be observable both by the sense of touch and sight. The variations include hardness or softness, roughness or smoothness, thickness or thinness, swelling and breaches such as wounds or ulcers. Under this heading of texture should be included various lesions such as papules, vesicles, pimples and pustules, and hypertrophy involving any of the layers of the skin, and often involving the subcutaneous tissues. (*c*) Sensibility. Alterations of sensibility may consist of reduced sensation (anæsthesia), increased sensitiveness (hyperæsthesia), itchiness (pruritus), and actual pain.

The term **dermatitis** is applied to inflammation of the skin or any part of it. It is, however, frequently restricted clinically to those conditions in which all the layers of the skin are involved.

When a lesion occurs in the skin in the course of a systemic affection, it is called an **exanthem,** and such a condition is not usually regarded as a skin disease proper, as in the case of variola, distemper, etc.

The *treatment* of skin diseases divides itself into constitutional and local treatment. Sometimes either method is effective, but in the majority of cases a combination of the two is advisable. The internal treatment is mainly devoted towards the restoration of healthy functions and normal vitality of the patient, which is often sufficient to bring about a restoration of the local skin affection. For this purpose it may be necessary to use aperients, to be followed by tonics, amongst the best of which for cutaneous affections are arsenic, iron, quinine, and silver (collargol), together with nutritives such as linseed for herbivora and cod liver oil for carnivora. Local remedies consist of sedatives, stimulants, emollients, refrigerants, astringents, antiseptics, and parasiticides, the choice depending upon the character of the symptoms exhibited and the cause of the condition. The following remedies may be found useful and should be generally employed in weak dilution: *Sedatives:* carbolic acid, acetate of lead, carbonate of zinc, belladonna, cocaine. *Stimulants:* zinc sulphate, zinc chloride, mercurial preparations, tar preparations, cantharides, and oil of rosemary. *Emollients:* various simple ointments with lard, lanoline, or vaseline as the basis, also weak alkalies. *Refrigerants:* evaporating lotions such as weak spirituous solutions, or the application of cold water; a freshly prepared solution of potassium nitrate and chloride of ammonia, or solution of potassium nitrate in vinegar and water. *Astringents:* tannin, chloride of zinc, weak solutions of metallic salts or of mineral acids. *Antiseptics:* carbolic acid, salicylic acid, chinosol, eusol, weak mercurial salts, iodine. *Parasiticides:* sulphur, tar preparations, iodine, salicylic acid, beta naphthol, and the various antiseptics in slightly stronger concentration. The use of these agents is indicated under the heading of the various skin diseases. It is impossible to lay too much stress upon the necessity for a proper and thorough application of whatever remedy is selected; the great proportion of the failures in the treatment of skin diseases in veterinary patients is due to improper application of dressings. The best remedy in the world will be absolutely useless unless properly applied. Careful selection is also necessary, since it is an undeniable fact that many skin affections are aggravated by improper external applications.

Erythema.—Erythema is a condition of active hyperæmia of the most superficial vascular layers of the skin. It is usually unassociated with any important structural change beyond occasionally some slight swelling and sometimes a little irritation. Erythema may be caused either by friction, pressure, bruising, by excessive heat (scalding or heat of the sun), or excessive cold. It may also be brought about by superficial irritants either chemical or bacterial. Examples of friction erythema are furnished by the rubbing together of two opposed surfaces of the skin, as in the case of thighs or armpits, when it is called **erythema intertrigo.** In cows a similar condition occurs by the chafing of the udder with the legs (**erythema mammillarum).** When the friction is due to a foreign body such as shafts, collar, or other harness, it is called **erythema paratrima. Secondary** or **symptomatic erythema** may result from dietetic errors, and is seen most frequently in white-skinned dogs, particularly under the abdomen, and is sometimes erroneously called **red mange.** It may occur in horses and cattle from feeding excessively on certain foods such as clovers, lucerne, distiller's grains, potatoes, and malt. Reddening of the skin may also occur in the course of various infectious diseases.

Symptoms.—The most important feature of erythema is a reddening of the skin, and therefore in animals is most easily recognized in those possessing white skin. The redness passes away with light pressure as by passing the finger over the part. There is sometimes a little heat and pain, occasionally some irritation. In those instances where there is extravasation into the deeper structures of the skin there may be some swelling as in collar and saddle scalds. The epithelium on the surface is slightly in excess and the skin may appear scurfy if the condition has lasted long, but there is never any accumulation of epithelium. In dietetic erythema in dogs the condition is usually painless, and is often associated with some irregularity of the bowels with a tendency to constipation.

Treatment.—Erythema rarely calls for drastic measures. Locally all that is required is some mild sedative astringent such as lead lotion or white lotion (zinci sulph. and plumbi acet. āā grs. x.-℥i.), or zinc oxide or zinc carbonate ointment in intertrigo. In paratrima the cause of the chafing must be removed and the skin may be hardened by tannic acid lotion (1-40). In symptomatic erythema a saline purgative may be given, to be followed by a course of tonics such as arsenic, quinine, gentian. A change of diet should be prescribed when dietetic origin is suspected.

Pruritus; Itching of the Skin.—Pruritus is a peculiar disturbance of sensation of the skin giving rise to the desire to rub or scratch the affected part. Pruritus may occur in many conditions without visible skin lesions. It may thus be due to chronic indigestion and also to chronic nephritis. It may also be brought about as the result of parasites in the alimentary tract. One frequently meets with pruritus in dogs in the absence of any other discoverable abnormality. The writer has frequently observed pruritus in the region of the loins and croup in dogs due to reflex irritation from inflamed anal glands. The condition known as "Scrapy" in sheep observed in Scotland is also a form of pruritus. Irritation of the skin due to external parasitic causes is not included under this heading.

Symptoms.—The irritation of the skin may be continuous or occur paroxysmally. The affected animal will lick or scratch or rub the affected part more or less severely, and may produce a scaly condition or thickening of the skin or sometimes even severe sores. Pruritus may lead to loss of condition owing to lack of rest and exhaustion.

Treatment.—Both local and internal treatment are necessary. Local treatment consists of the application of sedative dressings, amongst the best of which will be found a combination of liq. picis. carbonis and carbonate of zinc in ointment or lotion, or salicylic lotion; for localized patches menthol 1-10 or 20 in olive oil, or the local application of cocaine in lanoline 1 per cent. In each case the dressing should be applied with gentle friction. Internally temporary good results may be obtained by the administration of bromides, aspirin, or antipyrin. A mild purgative should be given, either saline or calomel, followed by a course of arsenic. In the case of dogs the writer has obtained excellent results from the hypodermic injection of 1 per cent solution of collargol, the dose being from c.c. ii.-v. This may be repeated a week later, and will usually afford excellent relief for five or six weeks, when it may be repeated if the symptoms recur.

Hyperidrosis (Excessive Sweating).—This is a condition of excessive moisture of the skin without any appreciable skin lesions. It is due to some functional disturbance of the sweat glands, frequently of nervous origin. It may also be symptomatic, as in acute rheumatism, tetanus, colic, and often in general debility. Excessive sweating may also occur in horses, due to exertion when not in hardened condition.

Symptoms.—There is profuse perspiration, which may involve the whole body or may be limited to localized patches. The affected patch may be sharply marked off from the dry skin. Sometimes one side of the body is affected, at other times the posterior half of the body. The latter frequently occurs in horses with broken back. There may be some alteration of sensation of the skin of affected part with partial anæsthesia or hyperæsthesia. In the case of horses affected there is general weakness with inability to do prolonged work, and frequently emaciation.

Treatment.—When symptomatic the cause must be discovered and treated. When it is due to local affection of the sweat glands, the application of refrigerants and astringents, such as lead lotion or white lotion or solution of tannic acid, may afford relief; while internally a course of arsenic or of potassium iodide may be given. In excessive sweating in young horses a full conditioning course must be prescribed, commencing with a purgative and light progressive exercise, together with an easily digested nutritious diet. Sometimes a course of tonics, particularly nux vomica, will be found serviceable.

Hæmatidrosis ; Hæmatopedesis ; Sanious Sweats ; Bloody Sweats.—This condition may occur as the result of hæmorrhage into the sweat glands, as the result of which blood becomes mixed with perspiration. It may also be brought about by the bites of insects or ticks, or be due to nematode worms in the skin (*Filaria multipapillosa*). Sanious perspiration may occur in various septicæmic affections such as purpura. (In the human being it is said to be produced by severe nervous impressions.)

Symptoms.—Small droplets of blood may be seen on the skin, and sometimes small hæmorrhagic nodules may be seen or felt. The mucous membranes also show blood markings at the same time. If the condition is very persistent, or has been in existence for long, it produces great nervous and general debility.

Treatment.—Externally refrigerants or astringents should be applied ; application of plenty of cold water to the part followed by white lotion or tannic acid solution. Internally quinine and iron should be given. Ergot may be administered as a styptic in bad cases.

Dosage.—See tables, pp. 544-6.

Urticaria ; Nettle Rash.—In urticaria small local areas of the skin become suddenly raised in the form of weals of varying sizes. They usually develop quickly and may disappear completely in a very short time. The condition is one in which the superficial vessels of the part become suddenly congested from some cause or other, bringing about a rapid transudation of serous liquid into the deep layers of the skin. It is in this way that a nettle-sting brings about a similar effect. In the case of animals coming in contact with numerous nettles, lesions of this character may occur over wide areas of the body and limbs. It may also be brought about by the bites of insects. Symptomatic urticaria may occur in the course of systemic affections such as purpura, dourine, and occasionally in strangles and influenza in the horse.

In cattle it is frequently produced by bursting a warble maggot when attempting to squeeze it out of the back. Urticaria may also be due in any species to dietetic errors such as feeding horses on a too highly nitrogenous diet.

Symptoms.—The condition is more frequently met with in young animals than adults, and is not uncommon in horses at rest in the stable still receiving their working diet. The lesions occur very suddenly, and are usually raised circumscribed areas, flat on the surface, and elastic on manipulation. They vary in size from the diameter of a pea up to several inches. Their distribution is quite irregular, and they may be few or may be very numerous, extending over the whole body. There is no exudation on to the surface of the skin and no appreciable alteration of the epithelium. There is rarely any irritation in veterinary patients unless due to stings by nettles or insects, thus differing from the condition in man. In cattle it is not infrequently met with in the region of the throat, face, and eyelids, sometimes involving the dorsum of the tongue, as in blaine or ting, causing dyspnœa or even asphyxia. The lesions may pass away in a few hours or may last several days. The condition is frequently recurrent.

Treatment.—Urticaria rarely calls for urgent measures except in cattle. The food should be changed and a laxative diet prescribed. Locally simple sedative applications may be used, such as lead lotion or white lotion, or when due to stings, vinegar and water, or ammonia, or permanganate of potash solution. Internally a mild laxative may be given, followed with an alterative. For horses and cattle a useful combination is sulphur sublimatum and potassium nitrate āā ʒii., antim. nigrum ʒi., ft. pulvis, one powder to be given night and morning in the food.

As a preventive measure in horses, with a day or two enforced rest the corn ration should be reduced and a laxative mash administered, together with an alterative powder as indicated above.

In cattle the treatment must be a little more energetic. First administer a saline purgative and follow with carbonate of ammonia and potassium nitrate, together with local applications as in the horse. If there is any dyspnœa owing to affection of the tongue, the latter should be grasped and drawn well forward. In rare cases it may be necessary to incise the dorsum of the tongue.

Urticaria in Pigs.—In swine urticaria occurs under many different conditions. Most of the conditions are symptomatic in origin, and result from indigestion, salt poisoning, swine erysipelas, or swine fever. The lesions occur usually behind the ears, inside the thighs and forearms, and also on the abdomen. Treatment depends on the type of animal and its bodily condition. If in a good condition slaughter is usually the best. If, on the other hand, the pig is more valuable for breeding purposes, a purgative should first be given, such as castor oil or Epsom salts or sulphur, in the food. This may be followed by small doses of hyposulphite of soda, also administered in the food. If the disease is due to one of the contagious diseases, the pig should be slaughtered.

Eczema.—Eczema is an inflammation of the superficial layers of the skin associated with hyperæmia, and the production of vesicles, which are usually small and discrete. Occasionally the vesicles are so numerous as to become confluent. The vesicles may be followed by pustules, and later by the formation of crusts or dandruff. Many inflammations of the skin have been included under the heading of Eczema, but the term is more correctly limited to the vesicular condition described above. The following excellent definition of eczema is quoted by Little from Bateman: "Eczema is an inflammation of the skin, non-contagious, often limited to a single region of the body, characterized at its beginning by very small vesicles, not prominent, usually closely juxtaposed, or actually confluent, terminating in the absorption of the fluid they contain, or in superficial excoriations, accompanied by a serous discharge to which succeed scaling, desquamation, or renewed vesicular eruptions of the same type."

All species of animals are susceptible to eczema, but it is most frequently met with in the dog and cat and the horse.

Causes.—The causes of eczema are not well known. No organism has been isolated as being generally responsible for the condition, and it must be regarded as a non-contagious disease. The writer is frequently met with the statement on the part of veterinary surgeons that they have in hand cases of eczema which have definitely proved to be contagious, and the only answer possible to such a statement is that there has been an error of diagnosis, and that the condition, though simulating eczema, is some other affection. In a number of such cases the writer has been able to demonstrate the presence of various acari, proving the mistaken diagnosis of eczema for acariasis. In a large proportion of cases eczema is due to internal causes and is not infrequently associated with indigestion. At other times it is a complication of renal disease. In each of these two cases effete matter is expelled from the body through the skin, causing irritation and setting up the lesion of eczema. At other times the affection is due to nervous causes, as in those instances where the lesions follow the course of nerves (herpes zoster) and in cases of defective innervation as seen in the hind legs of dogs with partial paraplegia following the nervous form of distemper.

Lesions resembling eczema are frequently due to external agents, such as dirt which is matted closely into the skin, washing with irritant soap which has not been thoroughly rinsed out. This is quite commonly the case with soft soap which has been rubbed into the skin in the opposite direction of the hair both in horses and in dogs. Lesions which are practically indistinguishable from eczema may also be produced by the presence of cutaneous parasites such as lice and fleas, ticks and harvest bugs. Chemical irritants may also cause irritation of the skin resembling eczema, but in the view of the writer such conditions and those produced by parasites should be excluded from the term.

There are several different manifestations of eczema; it may be **acute** or **chronic,** the latter frequently being due to the infection of the acute form by pyogenic organisms. **Dry eczema** or cutaneous catarrh is the form in which the amount of vesicular liquid is very small and is rapidly absorbed by the superficial epithelium which is thrown off in the form of small scales. When patches are circularly grouped and rather dry, the condition is *herpes circinatus.* **Moist eczema,** also called *herpes humidus* or humid tetter, is that form in which the vesicular liquid is more copious and oozes from the surface. This form usually occurs in isolated patches. When the vesicles become infected with pus-producing organisms the condition is called **eczema impetiginoides.** In horses a peculiar form is often met with affecting the white skin of the legs and face, and frequently limited to these areas in the particular patient. In dogs a peculiar form frequently occurs between the digits and pads on the solar aspect of the foot, and this is called **interdigital eczema.** When eczema is believed to be due to kidney troubles it is frequently called **urinous eczema,** and this form is usually associated with an odour of urine emanating from the skin.

Symptoms.—In the earliest stages eczema occurs as an erythematous eruption, and when this is closely observed it can usually be seen to be vesicular in character. There is a varying amount of irritation, although in uncomplicated cases there may be complete absence of itchiness, while at other times pruritus is very pronounced.

In **dry eczema** after the first day or two all that can be seen or felt are small dry elevations, which can be picked off and are seen to consist solely of dry epithelium. If the tip had been removed at an early stage a little clear serous liquid would have been seen to ooze. Manipulation of the lesion is not painful owing to the fact that the deeper structures are not involved. This form of eczema may affect very large areas of the body or it may be limited to small localized areas as of the lips (buccal or labial eczema), or round the eyes (orbital eczema).

In **moist eczema** the outpouring of vesicular liquid is excessive and oozes from the surface, causing matting of the hair with very great irritation of the skin. The matted part may become underrun with pus, and affected animals may bite or rub the part, causing a serious blemish. This form is most frequently seen on the backs of long-haired dogs and cats, and sometimes horses. The affected patches are generally about the size of the palm of the hand.

When eczema occurs in horses at the bend of the knee or hock or behind the pastern, it often assumes a scaly character, and if neglected may pass on to psoriasis and the formation of chronic ulcers, acutely painful and causing great lameness. As already stated, it is not uncommon for eczema to affect white legs or white patches of skin in horses. In these areas the primary erythematous condition is usually easily recognized. It has often been stated, and there may be some foundation in fact for the statement, that the lesion in such cases is often due to too frequent washing and imperfect drying of the skin, but in the writer's view this is by no means the only or principal cause of the condition. He has frequently observed it affecting white patches on the body which have not been subjected to washing, and he has also frequently seen it occurring in a single white leg which has not been subjected to any different treatment or management from its fellows. He is of opinion that the lesion in such cases is more likely to be due to a tropho-neurosis.

A form of eczema may also occur between the digits of cattle and sheep. In each instance it is probably due to irritation by dirt and caked manure. In some cases it may be painful and cause lameness.

The feet of dogs, as already stated, may be affected with eczema on the lower surface of the foot between the pads and digits. The condition is often very painful, causing acute lameness. The affected dog spends a considerable amount of time licking and nibbling the solar aspect of the foot. This condition should not be confused with the development of the so-called interdigital cysts or abscesses which appear on the upper aspect of the interdigital space. By many observers these lesions are thought to be secondary to interdigital eczema, but in the wide experience of the writer the two lesions have very rarely been co-existent. The skin of the scrotum is often affected with eczema in dogs, commencing with erythema and vesicle formation, and followed by a rather painful crustaceous stage, in which the dog may bite and rub himself and cause ulceration and œdema. He also violently resents examination and manipulation of the part. In herpes zoster or shingles the distribution of the lesion corresponds with the course of certain nerves such as the intercostal nerves or down the hind limb on the course of the sciatic nerve. Occasionally a similar condition occurs more diffusely in the lower part of the hind limbs of dogs affected with chorea or partial paraplegia following distemper. In each of these cases the lesions are essentially vesicular and usually very irritable. The affected animal bites and rubs and scratches the parts and may inflict serious injury upon himself.

In all cases of eczema where there is much pruritus there is general loss of condition due to lack of rest. There are rarely any other systemic symptoms except in the urinous form, when there may also be present symptoms of nephritis. In uncomplicated eczema recovery leaves no permanent mark.

Treatment.— True eczema calls for both internal and external treatment. In the first place a laxative should be given to clear out the alimentary tract, and this should be followed by a course of nerve and alimentary tonics. Of these in chronic cases arsenic will be found particularly useful (Fowler's Solution or Donovan's Solution) administered with or after food daily. Arsenic should never be given in the acute stages, as it then appears to rather accentuate the condition than otherwise. Other useful agents will be found in nux vomica, Easton's Syrup, iodide of iron, and in obstinate cases frequently calcium chloride will give relief. If irritation is very intense it may be necessary also to afford relief by means of bromides or morphia. Pruritus may also be relieved in dogs and cats by the hypodermic injection of collargol (cc. i.-v. of 1 per cent solution).

A complete change of diet will frequently be found serviceable. Any of the so-called "heating foods," such as clovers and highly nitrogenous grains, should be avoided in the larger animals and linseed mash substituted. In dogs, if the diet has been largely carnivorous, the meat should be stopped or reduced and farinaceous foods and milk substituted, and *vice versa*. A complete change is the essential feature.

The choice of external applications must depend upon the character of the lesions. If there is any external irritant present it must be eliminated, such as the presence of dirt, parasites, or, as is so often the case, soft soap. If the case is seen in the early erythematous stages a mild dusting powder will afford relief, such as a mixture in equal parts of zinc oxide or carbonate, boracic acid and kaolin, to which may be added chinosol 1 per cent. Bismuth carbonate, bismuth subgallate, and tannoform will often be found useful. If the surface, however, is very moist, powders are not so useful since they are apt to cake and cause considerable irritation. In moist eczema any matted hair must be carefully removed from the affected areas, and it may be necessary in order to do this to soak the patch well first with a solution of carbonate of potash or the lather of a non-irritant toilet soap. The part should then be disinfected, either with a weak solution of iodine 1 per cent, chinosol 1 in 500-1000, or eusol (hypochlorous acid ½ per cent). When the part is thus thoroughly cleansed, the following lotion will be found very serviceable :

℞
Zinci carbonatis
Zinci oxidi ãã ʒiv
Sol. picis carbon. ʒss.
Aquæ calcis ad ℥vi.
Fiat lotio.

Other solutions found useful include white lotion, chinosol lotion 1 in 500, mercurous oxide wash (black wash: calomel gr. xxx, lime water ʒx.). In the desquamating form and also where the crusts are firmly adherent, ointments or oily dressings will be found more serviceable. Of the former, zinc ointment or sulphur ointment are good, or, better still, a combination of the two with or without the addition of ichthyol, or sol. picis carbonis ʒss.-ʒi.; salicylic acid ointment 1-20; or in very obstinate cases

℞
Naphthol beta gr. lxxx.
Plumbi oleatis ʒi.
Adipis ʒii.
Ft. ung.

Owing to the toxic effects of naphthol this dressing must only be applied to limited areas and not repeated more than twice a week. An excellent application for large areas where there is much soreness and excoriation is the cremor zinci of St. Mary's Hospital Pharmacopœia:

℞
Zinci oxidi ʒi.
Lanolini ʒiii.
Ol. olivæ ad ʒi.
Aquæ calcis ʒi.
Ft. pasta.

If there is much irritation, carbolic acid m. x.-ʒi. may be added. (The carbolic acid is better omitted in the case of small dogs and cats on account of its great toxicity for them.) For orbital eczema unguentum hydrarg. oxid. flav. 1 per cent will be found useful; for scrotal eczema in the dog the hard scales should first be removed by an emollient dressing, and after a few days this may be replaced by the dusting powder as recommended earlier. For interdigital eczema in the dog the following practice has given excellent results. First wash the feet thoroughly with soap and water, removing all caked dirt and scabs, and thoroughly rinse and dry. Then soak the feet for 10-15 minutes in the following solution in a small jam jar or galley pot:

℞
Pulv. aluminis
Zinci sulph. āā ʒi.
Aquæ ʒvi.
Ft. lotio.

The feet should then be dried and zinc oxide ointment rubbed thoroughly in between the pads and bandages applied. The dog must be kept at rest while under treatment.

In chronic eczema good results are difficult to obtain. The naphthol ointment already mentioned will often bring about good results, as may also the repeated application of tincture of iodine or zinci chlor. gr. x.-ʒi.; while in the horse a mild blister of hydrarg. iod. rub. 1-12 or 16 may re-establish a healthy action of the skin in local areas.

When chronic eczema is due to derangement of the kidneys salines should also be prescribed internally, together with antiseptic diuretics, such as hexamine and sodium salicylate. Cod-liver oil may also be given with advantage.

Lichen.—This is a skin affection characterized by eruptions of a papular nature. The papules are usually discrete and multiple elevations, closely grouped together and often angular in outline, with a smooth surface. There is a marked increase of the epithelium of the part which remains on the surface in the form of small crusts. In rare cases the lesions may coalesce. If there is any exudate into the lesions it is small in quantity, and is absorbed by the epithelium covering it, so that the affected patches are usually dry. The condition is often indistinguishable from the papular stage of eczema. In the majority of cases the lesions are cold and painless in veterinary patients, and there is little or no itchiness. In those few cases where itchiness occurs the condition is called *prurigo*. Any animal may be affected, and it may occur in any part of the body, but it is most frequently met with in the horse, affecting the lips and face (*lichen labialis et facialis*). The lesion often becomes chronic.

Treatment.—Internally, administer an aperient and keep the bowels in a relaxed condition with salines, and follow with a course of arsenic, mercury, or sulphur. Alimentary tonics are also serviceable in many cases. If there is much irritation it may be necessary to administer potassium bromide or opiates.

Externally, soak parts with a hot solution of sodium carbonate and remove the softened scales; then paint the parts with tincture of iodine 1-4 or 6. Following this may be applied salicylic acid ointment (5 per cent), or zinc and sulphur ointment with ichthyol as recommended for the scaly form of eczema.

Pityriasis; Dandruff.—This is a superficial inflammation of the skin in which there is an abnormal production of epithelium, which is thrown off in the form of small scales. The causes of the condition are variable. In most cases dandruff is symptomatic, and in the veterinary patient is most frequently due to skin parasites, such as lice or mange acari. It may also occur in the course of digestive diseases, while in a large number of cases no appreciable cause can be discovered. The later scaly stage of eczema may readily be confused with it. Occasionally pityriasis is congenital, and is met with in newly born calves and foals and puppies.

Symptoms.—The condition may be met with affecting any part of the body, or it may extend over the entire body. The coat is usually dry and dull, and it contains a considerable quantity of small scales almost like dust, greyish-white in colour (furfuraceous). There may or may not be any irritation of the skin according to the origin of the condition. There are occasionally bald spots which may slowly extend. On examining the surface of the skin no other abnormality is generally discoverable, although in rare cases it may be slightly reddened.

Treatment.—Internally, keep the bowels in a relaxed condition, and administer a course of tonics such as arsenic and nux vomica. A change of diet should be prescribed avoiding leguminous foods. For herbivora boiled linseed is very useful, and a little cod-liver oil may be administered to carnivora. Locally, the skin may be soaked with a weak solution of carbonate of soda to which may be added a little creolin. If there is irritation it may be relieved with a weak salicylic ointment 1-20 or chinosol solution 1-500. Frequent and vigorous grooming and well-regulated exercise are essential.

Ichthyosis; Fish-skin Disease.—This is a condition of inflammation of the skin in which there is increased formation of epithelium, which becomes agglutinated and thrown off in the form of large dry horny scales, something like large fish scales. It is frequently congenital, and though it may occur in any species of animal it is most frequently met with in calves.

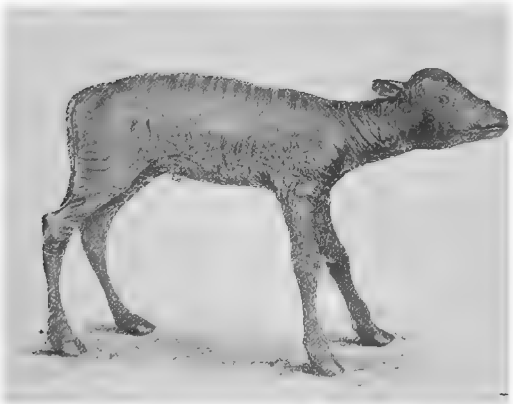


Fig. 80.—Congenital "fish-skin" disease.

Treatment.—Temporary improvement may be obtained by the use of the same remedies as recommended for pityriasis, the salicylic dressings being particularly useful. In congenital cases it is questionable whether the condition is worth treating. The internal administration of thyroid extract may be tried.

Psoriasis.—This is a condition of the skin in which considerable patches become inflamed, and on the surface of such patches there is a heaping up of epithelium which tends to accumulate where it is formed. The lesions form patches of varying size, and the surface is dry and dark grey in colour. In almost all cases in the lower animals when this condition is first observed it has a diffuse appearance, although in some instances it may commence in the form of papules which coalesce, thus producing the diffuse patch. It may be met with in all species, but is most common in horses and in cattle.

The cause of psoriasis is somewhat obscure, but there is reason to consider that heredity may play a more or less important part. In some instances it is probably parasitic in origin, while at other times it may be due to some micro-organism. It may also be brought about by friction, by such things as harness in horses and in working cattle.

The term psoriasis is also frequently applied in veterinary practice to the scaly form of eczema in which the crusts remain adherent.

Symptoms.—In psoriasis the superficial layers of the skin become inflamed, and an accumulation of greyish epithelium occurs. It often commences as an erythema, but it is seldom serious in effect unless implicating some parts in which considerable movement takes place, and particularly so in the bends of joints. It is not infrequently met with along the sides where the traces rub and also down the croup and quarters. When it affects the bends of joints the horny skin cracks and ulceration is set up with considerable pain, causing severe lameness and often indisposition to move. The commonest joints to be affected in the horse are the back of the knee, when it is called **psoriasis carpi** or **mallenders,** or in front of the hock—**psoriasis tarsi** or **sallenders.** A similar condition often occurs in the hollow of the pastern and may set up *cracked heels.* In these situations the condition is likely to cause knuckling over of the knees or fetlocks, both in the acute stages due to pain, and also in the chronic stages due to contraction of inflammatory connective tissue. When the heels are affected the horse may go on the toe, and as the leg is often somewhat swollen at the same time as the result of infection, a mistaken diagnosis of strain is not infrequently made. The disease has a tendency to become chronic.

Treatment.—It is often difficult to obtain good results. External remedies frequently suffice, but internal treatment may expedite recovery. A laxative should be given, followed by a course of alteratives such as arsenic, antimony, and sulphur. Locally, the epithelium should first be softened with a hot solution of carbonate of soda and much of the epithelium may then be removed. This should be followed with an ointment containing some antiseptic, the actual antiseptic being probably of less importance, unless ulceration has taken place, than the emollient itself.

Amongst the best dressings will be found salicylic acid ointment (1-16 or 20), zinc ointment (1-8), chrysarobin ointment (2 per cent in mild cases, 8 per cent or 10 per cent in obstinate cases), iodine ointment (5 per cent). Whatever ointment is used must be *very thoroughly* rubbed in.

In chronic cases in the bend of joints in horses, healthy action can often be produced by the application of a paste consisting of equal parts of crystalline acetate of lead and sulphate of zinc triturated together. It is usually unnecessary to add any vehicle, and the paste should be freshly prepared. If, however, it is intended to be kept for some time a little glycerine may be added to keep it moist. In other cases it may be necessary to lightly cauterize the chronic ulcers, and even to fix the joint by means of a splint or poroplastic felt. When the heels are affected, it is a good plan to put on a high-heeled shoe until healing is complete, after which the heel can be gradually reduced. It is sometimes asserted that this practice tends to produce a permanent contraction with knuckling and raising the heel from the ground. That, however, is not the experience of the writer. The practice undoubtedly expedites healing, and therefore reduces the amount of cicatricial tissue formed, the contraction of which is responsible for the permanent raising of the heel.

In obstinate cases in other regions the application of biniodide of mercury ointment 1-20 will frequently be found useful. In animals that are susceptible to this condition, psoriasis is often recurrent, and there is very little can be done to ward off attacks beyond avoiding the use of soft soap on the legs and at the bends of joints. The skin of the legs should also be thoroughly dried if it gets wet, flannel bandages being used for this purpose. Careful supervision should also be exercised, as early recognition and appropriate treatment will help to keep the condition well under control.

Mallenders and Sallenders. See " Psoriasis."

Cracked Heels. See " Psoriasis."

Scleroderma ; Hardness of the Skin.—This is a chronic dermatitis in which the skin becomes thickened, hard, and dry, and can no longer be picked up in folds. The cause is unknown, but in many cases it is probably parasitic. It may be met with in horses and asses, and it has also been described in swine. The writer has met with it affecting the neck of an ass in which the skin became almost board-like, extremely difficult to cut, and almost an inch thick. There was only slight increase of epithelium, the main thickening being in the skin substance. Microscopic examination of scrapings from the surface was negative ; prepared sections of the skin revealed the presence of a fungus, indistinguishable from the *Penicillium glaucum,* in the deep layers of the skin. Cultures were made, but efforts to reproduce the condition with the culture were negative. The position of the fungus, however, appeared to indicate an intimate connection with the causation of the condition.

Treatment of this condition is hopeless.

Elephantiasis ; Pachydermia.—This is a condition of inflammation of the skin and subcutaneous connective tissue most commonly met with in horses. It is brought about by chronic or frequently repeated irritation, and often originates with lymphangitis (see p. 36) with an increase of connective tissue round the lymphatics of the part. In these latter cases it usually affects a limb. Any part of the skin, however, may be affected. When there is a heaping up of epithelium, the term pachydermia is usually given, while in elephantiasis proper the thickening is deeper and the epithelium is not much increased in quantity. A limb affected with elephantiasis may become enormously thickened, and flexion of the joints is difficult or impossible. The condition is quite painless, and regular exercise and work tend to check the progress of the condition.

Treatment.—Internally an aperient should first be administered, and the bowels should be maintained in a more or less relaxed condition. A course of arsenic or of potassium iodide may be given, or biniodide of mercury may be substituted for the latter. Externally, if the skin is scaly it should first be soaked with an alkaline solution and then dressed with an antiseptic astringent such as lead lotion, white lotion, or chloride of zinc (2 to 4 per cent). Unless seen in the early stages, treatment is rarely successful beyond checking the progress of the condition. Regular exercise is essential, and affected horses are better at work since the condition is painless, and even one day in the stable may be followed by an increased swelling of the limb.

Alopecia ; Baldness.—This is a condition in which there is loss of hair from a part normally covered with it. All animals are susceptible to the condition. In the majority of cases it is due to a disease of the hair follicles, and this may be brought about by interference with nutrition of the skin (tropho-neurosis), general debility, or it may be due to bacteria or to animal parasites. Cicatricial alopecia may also be met with in localized patches due to mechanical or other injury of the skin, resulting in destruction of the hair follicles. In rare cases the condition is congenital, affecting almost the entire body surface in foals, calves, and dogs. When baldness occurs in circumscribed, more or less circular areas, it is called *Alopecia areata.*

This form is sometimes due to nervous influence; at other times it is undoubtedly due to ringworm. Baldness may be due to toxic causes and may thus be met with in various contagious diseases such as purpura, strangles, influenza, distemper, and swine-fever. Baldness is also frequently met with in pregnant animals, and in these cases the cause is entirely unknown.

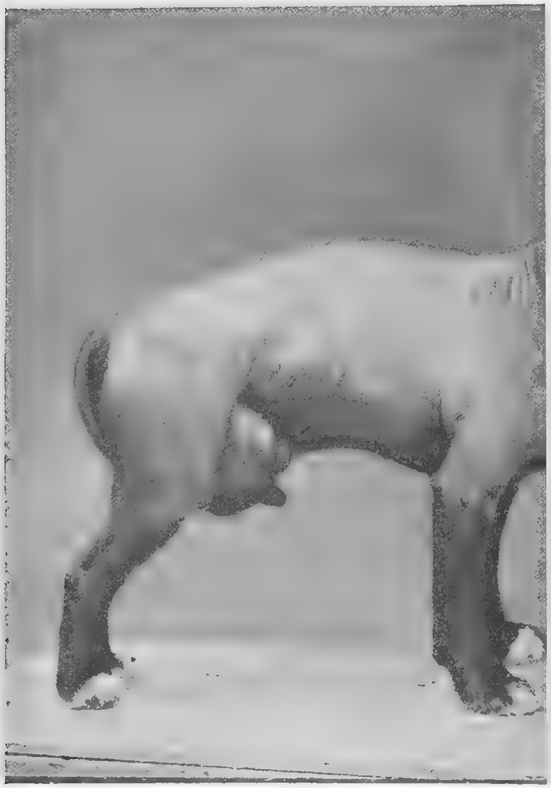


Fig. 81.—Alopecia and carcinoma of a testicle.

In dogs it is said to result from in-and-in breeding, particularly in toy black-and-tan (Manchester) terriers. In many of the latter cases, however, the writer has frequently been able to demonstrate the presence of *Demodex folliculorum*, and on making enquiries in almost every case he has ascertained the fact that the puppies were born with a good coat, and that the loss of hair commenced some weeks, and sometimes months, after birth. He is therefore sceptical as to in-and-in breeding being a cause of alopecia.

Treatment.—The treatment depends upon the cause of the condition. When symptomatic, the primary disease must be attended to, and as the animal gets stronger the coat gradually returns. In other cases the skin should be well massaged daily and a stimulating antiseptic lotion well rubbed in. Good results will frequently be obtained by the use of chinosol solution 1 in 500, weak iodine solution 1 per cent, paraffin, salicylic acid in ointment or lotion. The addition of a cutaneous stimulant, such as tincture of cantharides or oil of rosemary, expedites the growth of hair. The following will be found a useful hair stimulant:

℞
Acid salicylici ʒss.
Spt. vini rect. ℥iii.
Tinct. canthar. ℥ss.
Ol. rosmarini ʒi.
Ol. ricini ad ℥viii.
Fiat lotio.
Sig.—To be applied daily with friction.

In the case of horses a mild blister of cantharides or biniodide of mercury will stimulate the growth of hair. Internally a course of nerve and general tonics is good, such as arsenic, strychnine, and phosphates. In horses and ruminants with extensive alopecia the use of boiled linseed internally appears to exert a beneficial effect, and in carnivores cod-liver oil is similarly useful. In cicatricial alopecia treatment is obviously useless, since the hair follicles are destroyed.

Ecthyma.—Ecthyma is a localized inflammation of the skin characterized by the formation of pustules, which may be superficial in character, or deep-seated and result in some loss of structure. Ecthyma may be conveniently described under two headings, *Acne* and *Impetigo*.

Acne; Pimples; Boils; Furunculus.—Acne is a folliculitis of the skin, *i.e.* an inflammation of the hair follicles or the sebaceous glands, resulting in the formation of pustules. The lesions are circumscribed and may be comparatively superficial (*pimples*), or they may be deep-seated, involving the deeper structures of the skin. When these latter lesions are single or isolated they are called *boils*; when they are small and appearing in crops they are called *furuncles*. Acne is generally due to the entrance of pyogenic organisms which have gained access through the orifices of the skin, or into the hair follicles, as the result of rubbing. In fact it may be regarded as a "rubbed-in" disease; consequently it is met with mainly in those parts of the body which are exposed to rubbing by harness and other objects. It is frequently observed a few days after clipping, probably owing to scratching with the machines. In this way it may be met with as a complication in strangles in horses, infective discharge being spread over the skin in grooming. It may also be brought about by the choking-up of the ducts of sebaceous glands. A lesion, practically indistinguishable from acne, may also be caused by biting flies. Another smaller pustular eruption may be produced by the application of certain medicaments, such as iodine, tar, vaseline, and other chemical irritants. In these instances the lesions are variously due to choking up of the glandular ducts, or to chemical irritation, or both. The lesion set up by the invasion of the skin by the parasite *Demodex folliculorum*, accompanied by staphylococci in any species, is a form of acne. Acne appears to be more frequent in the spring and summer than in the cold weather. All species of animals are susceptible, but it is most frequently met with in horses, sheep, and dogs.

Symptoms.—The lesions may occur in any part of the body, but, as already indicated, they are most likely, in horses, to be met with under the collar, saddle, or along the sides, or at any place that may be rubbed by the harness. In the dog the lesions are frequent about the head and under the abdomen, while in sheep they are principally found inside the elbows and thighs, where the wool is scarce and the skin is thin.

In simple acne (pimples) the pustules are very small and rarely exceed the size of a lentil. If examined early the lesions may be felt as small elevations which in a day or two become pointed and contain a small amount of pus-like material. They are usually numerous and are painful, but not invariably so, and the skin carrying them can be freely moved about over the subcutaneous tissues.

In the more severe form (furuncles and boils) the lesions are deeper seated and may involve the whole structure of the skin. They are hot and painful, and surrounded by a zone of hyperæmia. The affected animal resents manipulation of the part, and horses will often cringe away when the part is approached, even before it is touched. The lesion cannot usually be moved so freely on the underlying structures. When mature it has a yellow point in the centre which bursts, eliminating pus and sometimes a small necrotic core. At other times the painful lesion appears to subside without pointing or bursting (blind boil). Boils or furuncles under the collar in a horse may cause the animal to become a jibber.

The duration of the lesions depends upon the depth of skin involved. In the superficial form it may only last a few days or a week, while in the deep-seated form it may be two or three weeks before recovery occurs. During this time other lesions may crop up so that the affection may become somewhat prolonged. Healing in the deep-seated form leaves a more or less permanent mark, which may be attended with loss of hair or change of colour.

In folliculitis due to blocking of the sebaceous glands, an accumulation of curdy material takes place and causes a local swelling which varies from the size of a pea to a pullet's egg. These are called *sebaceous cysts* or *comedones*. The walls of these cysts may actually degenerate and undergo calcification.

Except in very severe cases acne does not set up systemic symptoms.

Contagious Acne.—A variety of acne in the horse, undoubtedly contagious, is due to the Preisz-Nocard bacillus, and is often called American skin disease. This also occurs in two forms — superficial and deep. In the former the lesions are very numerous, occurring chiefly on the shoulders and sides. They are very superficial and practically painless, and do not leave any permanent marking. There is a great disposition to spread all over the surface of the body of the affected horse, and to other horses, by means of grooming utensils, rugs, harness, etc. This affection is said to have been introduced into England with Canadian horses in 1879, but it was already in existence on the continent of Europe before that date. An outbreak of a much more severe form of contagious acne occurred in Great Britain in 1913–1914, and was also said to have been introduced from the American continent. In this form the lesions were less numerous but more deep-seated, very painful, and not nearly so amenable to treatment. The writer observed numerous cases in which the affection lasted for months, and not infrequently affected animals had to be destroyed.

In *sheep* the lesions of ecthyma occur commonly in the parts where there is very little wool, such as inside the thighs and elbows. They are pointed and contain pus, but, unless very numerous, cause very little systemic disturbance. Occasionally lesions of acne are met with along the middle of the back, and the fleece of that part is ragged and torn. This is not infrequently due to the use of a too irritant sheep dip, and care should be taken to avoid confusing it with sheep-scab. It is common to find several sheep affected simultaneously, but there is no direct evidence of contagion.

In **swine** the lesions of acne are usually black spots, and are due to *Demodex folliculorum*, or to in-turned hairs.

Treatment of Acne in Horses.—In the superficial diffuse form the skin should first be soaked with a hot solution of sodium carbonate, which softens the epithelium and allows the subsequent dressings to penetrate better. The condition then yields readily to the application of simple disinfectants such as: creolin solution 2 per cent; oil of tar ʒi. in rape oil Os̄s̄.; salicylic ointment 1 in 20; or sulphur liniment (sulph. sub. ʒii, potass. carb. ʒi., ol. picis ʒi., ol. rapæ Os̄s̄.). In the more severe forms, as in boils, local pain must be reduced by hot fomentations, such as hot water or antiseptic poultices, and when mature the boil should be lanced with a crucial incision and its contents—pus or necrotic core—removed. It must then be disinfected with a strong disinfectant as a first dressing, such as pure carbolic acid or tincture of iodine, and subsequently treated as a simple wound. The carbolic acid is very conveniently applied by dipping a probe or small grooved director into it and inserting the point into the centre of the lesion. At other times the application of biniodide of mercury ointment is effective in these persistent cases. It frequently happens in horses that after such a lesion has completely healed there remains some irritability of the part for a considerable time, such as may actually cause lameness, especially if under the collar. In the severe form of contagious acne good results are very difficult to obtain. In such cases an autogenous vaccine may be tried. Internally, a course of tonics should be prescribed, such as arsenic, sulphur, iron, strychnine, or quinine. Thorough disinfection of harness, clothing, and grooming utensils should be carried out, and also the stable and any places where the animal may have rubbed (shafts, etc.). When the contagious variety is suspected, isolation should be practised. Affected horses should not be worked, but daily exercise should be given.

In dogs the treatment of the condition is very similar to that of horses, but the tar preparations must be used with caution owing to their toxicity for dogs. Salicylic preparations (1-20) are very serviceable, as also are chinosol (1 in 500), and eusol (0·5 per cent hypochlorous acid). Internally, a course of tonics should be prescribed such as arsenic, or cod-liver oil, malt, and iron mixture. The acne lesions of follicular mange in dogs generally yield readily to autogenous vaccines.

In all species, small impacted sebaceous glands may be relieved by puncture and squeezing, but the larger ones should be dissected out, otherwise they are likely to recur.

In sheep with acne, a local application of carbolic lotion or sulphur dressings should be resorted to, and internally a small dose of saline administered and followed by a course cf tonics.

Impetigo.—Impetigo is an eruption of the skin consisting of a superficial pustule, often accompanied by a surrounding zone of redness or hyperæmia. The lesions are usually painless and often termed "cold pustules" in contradistinction to acne, the lesions of which are often called "hot pustules." The lesions in impetigo are always due to a micrococcus, but the condition is generally met with in animals with reduced vitality from various causes. It may thus be met with in puppies at the time of teething, and also the subjects of worms. In bitches it commonly occurs about the time of parturition and when rearing puppies. A lesion practically indistinguishable from impetigo is also met with in the so-called cutaneous form of distemper. In milch cattle impetigo is frequently met with, affecting the skin of the udder immediately after calving, particularly in highly fed cows. This form often assumes a contagious character, and has often been mistaken for vaccinia. Impetigo is also met with occasionally in horses and in swine, but more rarely than in dogs and cattle. By some authorities impetigo is regarded somewhat questionably as the pustular form of eczema (*E. impetiginoides*).

Symptoms.—The lesion consists of a very superficial flat pustule which may be preceded by a tiny vesicle under the epidermis. The surface is yellow and soft. Generally the lesions are circular and discrete, and from one quarter of an inch to half an inch in diameter, but if numerous they may be confluent. They are usually free from irritation, and are often surrounded by a red zone. They rupture quickly and form yellow crusts, which soon fall off. Sometimes the hair over the part is matted, as the result of which irritation may arise. When the lesion occurs on the hairless parts, it frequently leaves, on bursting, a simple ragged edge of epithelium, and the previous existence of the pustule may be indicated by a spot which is slightly cleaner than the surrounding area, and the margin of which appears as though etched out. Impetigo always runs a rapid course, and this fact, together with the absence of irritation, serves to distinguish it from eczema.

Treatment.—Impetigo is amenable to very simple measures. Internally a course of tonics should be prescribed, while locally the scabs should be removed by fomentation with a mild antiseptic and astringent lotion, such as chinosol 1 in 500, eusol, carbolic acid 1 per cent, creolin 1 per cent. Subsequently a mild dusting powder, as boracic acid and starch, may be applied. In cows the coal-tar preparations and eusol should be avoided owing to the danger of tainting the milk; chinosol lotion or boracic lotion are preferable.

Pemphigus.—Pemphigus is a superficial inflammation of the skin characterized by the development of large vesicles or bullæ. It is met with in the horse and ox, and occasionally in the dog, but in any case must be regarded as a rather rare skin disease. It usually runs a benign course. Its cause is unknown.

Symptoms.—The lesions may be met with in the horse on the neck, chest wall, and lower part of the abdomen, and in cattle they are most often seen in the region of the loins and the hind limbs. They may, however, be met with in any part of the body. There is first a slight swelling of the skin, on which there develop vesicles which continue to increase in size for two or three days, and in that time they become as large as a hen's egg. They contain a clear yellowish liquid which sometimes becomes turbid. As a rule, there is no irritation of the part, but sometimes intense irritation is set up, causing rubbing and scratching. When a vesicle bursts, a red, angry-looking base is exposed which becomes covered in a few days with a fine pellicle of epithelium. Until this has occurred a moisture continues to exude from the surface. On bursting, the wall of the vesicle may remain attached in the form of a scab, or it may become detached by rubbing in irritable cases. Recovery is usually complete in from two to three weeks. The attack is occasionally accompanied by slight fever and irregularity of the bowels, but in the majority of cases there is no systemic disturbance.

In rare cases pemphigus assumes a chronic course, the lesions being numerous and spreading extensively over the body. They show little or no tendency to heal, and set up emaciation, irregular bowels with a tendency to diarrhœa, and ultimately prove fatal from exhaustion.

Treatment.—In the early stages the lesions should be dressed with a mild antiseptic and astringent lotion such as chinosol lotion or lead lotion. If the lesion has burst it should be kept clean, and protected by mild dusting-powders such as boric acid and starch, or tannoform in boracic acid 1 to 8 or 10; subgallate of bismuth is also useful. If scabs form they must be removed after a thorough soaking with hot soda solution or soap lather. Internally a laxative such as sulphate of magnesia should be given, and a course of arsenic prescribed. The diet should be laxative in character.

Seborrhœa; Stearrhœa; Grease.—Grease is a form of chronic dermatitis of horses' legs in the neighbourhood of the fetlocks and pasterns. The inflammatory changes involve several layers of the skin, particularly round about the sebaceous glands.

Causes.—The cause of grease is still obscure. The condition is, however, most frequently met with in dark and badly kept stables, but it may also be met with in horses kept under good conditions. The discharge from the affected parts frequently contains a spirillum, but it has not been satisfactorily shown that this organism has anything to do with the etiology of the condition. The same applies to the occasional presence of a symbiotic acarus. The condition is, however, most frequently met with in horses with comparatively poor cutaneous circulation, as the result of which they have puffy legs (gummy-legged horses).

Symptoms.—The first change noticed is usually an increased production of sebum, which exudes on to the surface of the skin and has an extremely offensive penetrating odour. The leg is wet and the hair frequently stands out. There is at the same time a rapid increase in the production of epithelium, which also degenerates and becomes fetid. As the condition advances there is a marked increase in the connective tissue of the part, causing thickening of the skin of the leg. The skin papillæ also become considerably enlarged and develop into fungating wart-like growths, which are sometimes called "grapes." It has a tendency to become chronic and progressive. It is most frequently met with in the hind limbs, though occasionally it is seen in the fore limbs. In acute cases it may set up severe lameness and render animals unfit for work. In the less acute cases, however, affected horses are better at work, otherwise the whole limb becomes considerably swollen and the condition aggravated. In the majority of cases grease is painless. It is probable that there is some association of this condition with canker of the horses' foot.

Treatment.—The hair should first be removed from the affected parts and the leg thoroughly washed with hard soap and water containing washing soda. At the same time creolin should be added to reduce the offensive odour. After drying, the skin should be soaked with a strong disinfectant and astringent such as copper sulphate, zinc sulphate, and alum āā grs. xv. to ℥i., or zinc chloride 5 per cent solution, or corrosive sublimate 1 in 500 dissolved with a little hydrochloric acid. These lotions should be well rubbed in by means of a stiff brush. Cotton-wool may then be saturated in the solution and applied by bandage. After a few days of this dressing, salicylic acid, either in lotion or ointment, 5-10 per cent, should be well rubbed in. When there are vegetations the projections should be removed by the actual cautery—as they are so vascular there would be excessive hæmorrhage if removed by the knife. When the acute stage has been controlled, the condition may be kept in check by a daily application of 5 per cent zinc chloride solution to the whole area, and dusting the granulating or ulcerating patches with salicylic acid i., oxide and carbonate of zinc āā vi. Internal treatment should also be adopted, giving a course of arsenic and antimony. The hypodermic use of atoxyl has also been recommended, but the writer has not met with encouraging results from its use.

Erysipelas.—Erysipelas is a diffuse inflammation of the skin tending to spread indefinitely by means of the lymph spaces and lymphatic vessels of the affected part. True erysipelas as occurring in the human subject is due to a specific streptococcus. It is doubtful, however, whether this organism is pathogenic to the lower animals, and it is therefore questionable whether true erysipelas ever occurs in them. On the other hand, however, one may meet with inflammation of the skin with characters somewhat resembling erysipelas in man, and although the same term may be conveniently applied to these conditions, it would be better to describe them as *erysipelatous dermatitis*. Dermatitis of this character is sometimes met with in horses, sheep, and swine, and occasionally dogs, particularly affecting the head, neck, and limbs.

The causes are varied. Inflammations of this character may follow on injuries of the skin accompanied by infection with pyogenic organisms and fungi. It has also been stated that the feeding of horses on various materials will produce it, such as buckwheat, lucerne, etc.

Symptoms.—Erysipelatous dermatitis usually commences with a more or less painful area of the skin which appears swollen and deep red in colour. There may be several patches, which, if close together, may become confluent. The edge of the patch is felt as a raised ledge—the so-called spreading edge of erysipelas. In many cases vesicles and blebs form on the surface, and these may burst and form crusts. Other symptoms will depend upon the seat of the lesion. When occurring on the face they may interfere with feeding, or they may cause partial closure of the nostrils or orbits. If they occur on the limbs in the region of joints they will set up lameness, both from pain and mechanical interference with movement of the joint.

In a case in a dog observed by the writer, all four feet were affected. They became very considerably enlarged and swollen so that the dog could scarcely move about. It lasted for six or seven months, during which time numerous small vesicles formed and burst and healed, to be followed by similar lesions in close proximity, limited patches also occurring on one forearm and the nose; recovery ultimately occurred in all but one foot in which the condition became chronic. Systemic symptoms of an intermittent character are sometimes met with and consist of slight fever, inappetence, and general depression.

Treatment.—Local measures are of principal importance. They consist of the application of antiseptics and astringents such as lead lotion, zinc chloride lotion, carbolic acid, and eusol. A saturated solution of magnesium sulphate is sometimes useful. These dressings may be followed by application of a dusting-powder such as boracic acid and tannoform 1-8, or bismuth subgallate. In the case of the dog above referred to, numerous agents were resorted to from time to time, and each given a fair trial with very little beneficial effect, until acriflavine 1 in 1000 was used. This was rapidly followed by marked improvement in the case of three feet, while the fourth improved more slowly. Internal treatment should be prescribed in the form of tonics such as quinine and iron, cod-liver oil, etc.

G. H. W.

Parasitic Diseases of the Skin

Nematodes of the Skin and subcutaneous tissues

A number of round worms may be found parasitic in the skin or in the subcutaneous connective tissues of the horse, ox, and dog. Most of them belong to the family Filaridæ.

Filaria multipapillosa (*F. hæmorrhagica*).—This parasite is met with on the Continent of Europe affecting horses, mostly Russian and Hungarian. It sets up a condition known as "Parasitic skin-bleeding," or a form of "hæmatidrosis."

F. multipapillosa is a fine threadworm from 1 inch long in the male to 2½ inches in the female, carrying on its head a number of papilla-like markings, hence its name.

Symptoms.—Parasitic skin-bleeding is seen usually in the spring and early summer, and may continue intermittently for two or three months of the year. It is often recurrent for two or three years in succession before finally disappearing. Sweating and exposure to warm sun bring out the lesions, while cold and rest tend to check their development. The lesions are met with especially about the neck, shoulders, withers, and back, and at first consist of small, round, painless elevations of the skin varying in size from a pea to a hazel-nut. The hair soon becomes matted with exuding blood, which may also trickle round the spot. This is due to bursting of the nodule through a very fine puncture. The swellings are usually multiple and subside on bursting, but others take their place. They rarely give rise to constitutional symptoms, but they are apt to interfere with harness and prevent the horse being worked.

To find the parasite shave the affected part, look out for the development of a new lesion, and incise it just before it appears likely to burst. Even then it is sometimes difficult to find, as after puncturing the skin it quickly shrinks back into the subcutaneous tissues, from whence it may set up new lesions. The life-history of the parasite is unknown.

Treatment.—The affected parts should be clipped and washed and cooling lotions applied, such as weak acetic acid or spirituous solutions and antiseptics. The external application of the usual parasiticides is apparently of no avail. A course of arsenic may be prescribed internally. Care must be taken that harness, etc., does not chafe the lesions, which sooner or later tend to disappear spontaneously.

Filaria irritans (*Dermofilaria irritans*).—This parasite is met with affecting the skin of horses and setting up the condition known as "Granular Dermatitis" or "Summer Sores." The condition is met with principally in hot countries, notably Southern France, Italy, and Algeria.

The parasite in the skin consists of a small white threadworm up to $\frac{1}{8}$ inch in length, and is the larval stage of a parasite whose identity is still obscure. Roger considers it to be the larva of the oxyuris.

Symptoms.—The lesions set up consist of granulations affecting surface wounds or abrasions, with a tendency to spread. They vary in size up to a pea, and are soft, pulpy, and nodular, and may be found on the skin of any part of the body, but particularly on the legs and where the harness has rubbed. In the centre of the granulations may often be found a small caseous mass in the middle of which the small white worm may be found. The nodules can often be shelled out as though encapsuled, and may extend through the entire thickness of the skin. The lesions are very irritating, and the affected horse rubs the part, increasing the injury to the skin and extending the lesion. The condition persists till the end of the summer and sometimes longer. It ultimately heals but is liable to recur in successive years. The method of invasion of the parasite is not definitely known, but it is believed to be an external infection.

Treatment.—The treatment of this condition is not very successful. If the lesions are very limited excision is probably the best practice, or the application of the actual cautery. In more extensive cases the application of sulphide of arsenic in a thin layer has been recommended and also iodoform in ether or chloroform or collodion; or carbolic acid or orthoform in collodion 1 to 8. These agents are both parasiticides and local anæsthetics, and thus reduce the irritation and rubbing.

Cutaneous Nematodes in the Dog.—Small nematodes have been met with in small, discrete pustules occurring on the limbs of dogs. The condition in cases described by Möller and others lasted for several weeks, but as a rule yielded to general cleanliness and mercurial ointment.

Embryos of *F. immitis*, the mature stage of which inhabits the right side of the heart, have been often met with in the skin and subcutaneous connective tissues, either free or enclosed in small cysts.

Dracunculus medinensis (*Filaria medinensis*).—This parasite, which is also known as Dragonneau or Guinea-worm, sets up the condition known as Dracontiasis. It is met with in dogs and horses, and also the human being, particularly in the East—India, Persia, Egypt, round the African Coast—and also in some parts of South America.

The parasite is about $\frac{1}{16}$ inch thick and ranges from 15 to 30 inches long in the female and $1\frac{1}{2}$ inch in the male. The female is found in the subcutaneous tissues chiefly about the limbs, where it causes an extremely painful vesicle about the size of a haricot bean. Sometimes there are several parasites. On bursting, a small amount of pus-like material consisting chiefly of embryos exudes, and the head of the worm then protrudes. It may later be extracted by gentle firm tension, such as may be exerted by winding it on a match-stick. If the worm is broken in the attempt to extract it, it recedes into the connective tissues and gives rise to a painful abscess.

Life-history.—The young larvæ which are liberated from the female find their way into fresh water and there are swallowed by small crustaceans (various cyclops). In this host they undergo development requiring several weeks. The intermediate hosts are then taken up in drinking water by animals or man. In the stomach of the new host they are set free by the gastric juice. They then penetrate the wall of the stomach or intestine and migrate into the subcutaneous connective tissue, where they attain sexual maturity in about nine months; fertilization then occurs and the males perish.

Treatment.—An attempt should first be made to extricate the worm as indicated, on a match-stick. If that fails it can sometimes be removed by incising the track of the parasite. If an abscess has formed, the usual treatment for abscess should be adopted.

Preventive measures consist of straining or filtering all drinking water, or allowing animals to drink only through gauze muzzles.

G. H. W.

Ringworm; Tinea; Dermatomycosis

Ringworm is a contagious disease of the skin caused by a mould or fungus belonging to the class Hyphomycetes and classed in the family Trichophyta. The typical lesion consists of a circumscribed elevated patch which becomes covered with scabs or scales, and may or may not be denuded of hair.

The Trichophyta include several groups of parasites, viz.:

(*a*) *Trichophyton megalosporon.*
(*b*) *T. microsporon.*
(*c*) *Achorion.*
(*d*) *Oospora.*
(*e*) *Eidemella.*

(*a*) *T. megalosporon*, spoken of simply as *Trichophyton*, is sometimes subdivided into classes according to the position of the fungus in the hair. When found exclusively on the surface of the hair-roots it is called *T. ectothrix*; when occurring within the hair-sheath, *T. endothrix*; when in both places, *T. endoectothrix*. *T. mentagrophytes* invades the hair follicles and may also cause the formation of pustules. *Trichophyton* sets up the condition known as *trichophytosis*, the lesion being called *Tinea tonsurans.*

(*b*) *T. microsporon*, generally spoken of simply as *microsporon*, sets up *microsporosis.*

(*c*) *Achorion* and (*d*) *Oospora* set up *favus ringworm* or *Tinea favosa.*

It has been suggested that the various ringworm parasites may be saprophytes and capable of independent existence apart from animal hosts, but all the clinical evidence goes to show that the great majority at any rate are obligatory and that ringworm can only be spread by contagion—mediate or immediate—from an infected animal.

Diagnosis of ringworm can generally be made by naked-eye examination of the characteristic lesions, but in some cases a certain diagnosis is only possible by microscopic examination and identifying the mycelial spores in the crusts or hairs. For this purpose material should be taken with a knife or curette from the margin of the lesion, together with some of the hairs of the part. This material should be soaked in a 10 per cent solution of caustic potash, heated, and teazed out. Several of the separate hairs with their roots should then be taken out and examined under the microscope under a cover-glass without staining, with a fairly high power ($\frac{1}{6}$th objective). If the condition is ringworm, the so-called spores will be visible in or around the root of the hair. The hair itself is usually frayed or split at its free end. If desired, a similar specimen of hair may be stained by the Gram-Weigert method to demonstrate the spores more clearly. Good preparations can also be made by cutting paraffin sections of the hair obliquely and staining by the Claudius method.

In the case of favus ringworm the fungus is very easily demonstrated by teazing out a little of the dry crust and soaking it in 10 per cent caustic potash solution and examining unstained.

Contagion of Ringworm.—Infection usually results from immediate contact of healthy with affected animals, or it may also occur in stables, sheds, or pastures through the medium of posts or other obstacles on which affected animals have rubbed themselves. It may also be spread by the sexual contact, and from parents to their suckling progeny. Harness, clothing, and grooming utensils are amongst the most important agents for spreading the disease in horses. The parasites are very resistant to external influence and may remain infective under favourable conditions for six months and probably longer. Infection is most likely to occur if the fungus falls on to an abraded skin.

Some of the forms of ringworm are transmissible from one species to another, and these characters will be noted under their respective headings.

Predisposing Causes of Ringworm.—It is a well-known fact that general neglect and dirty condition of the skin and surroundings predispose to attacks with ringworm. The same also applies to general debility resulting from malnutrition. The careless use of grooming utensils, especially curry-combs, predisposes, both as the result of conveying the parasite and the scarification of the skin. Youth must also be regarded as a predisposing cause to ringworm, since the condition is so much more frequently met with in young animals than in adults. The indiscriminate interchange of harness and clothing facilitates the spread, and the presence of such vermin as rats and mice predisposes to the infection of cats with favus. The period of incubation of ringworm varies from one to three weeks or a month.

Ringworm in the Horse.—Two forms of ringworm are met with in equines, viz. trichophytosis and microsporosis.

Trichophytosis.—Four different species of Trichophyton have been described in the horse, setting up lesions practically indistinguishable at any rate in three instances. The lesions are met with generally in the head, neck, and withers, and sometimes at the root of the tail. The hair first becomes tufted and matted and breaks off, the ends being very much frayed. There then develop raised circumscribed patches, at first small, but extending until they attain the size of a five-shilling piece. At that point growth usually ceases; greyish-white crusts of varying thickness are then produced, and greyish or yellowish scales may become detached. If hairs are pulled out from the edge of the lesion and their roots carefully examined, they will even be found to carry a little whitish powdery mass which on microscopic examination proves to be fungus. From the original patch the condition is spread by grooming or by rubbing, so that within a week or two of the first appearance of a lesion ringworms may be met with all over the body. If the scabs are removed, the patch underneath is bare of hair, smooth, and pinkish-grey in colour. There is rarely much irritation in this form. With one species, *T. mentagrophytes*, the hair follicles become affected, and when the scabs are pulled off small pustules may be seen. There is a certain amount of irritation in this form, and sometimes when the hair grows again it may be found changed in colour. Trichophytosis in horses is usually benign, setting up no systemic effects, and usually disappears in five to six weeks, although in the meantime fresh lesions may be set up, especially under dirty conditions and as the result of malnutrition and exposure, so that it may become almost chronic and difficult to control.

Microsporosis.—This form is met with in horses of all ages but particularly in foals. It affects various parts of the trunk, and when affecting adults is seen especially under the harness, saddles, etc. On the affected patch the hair becomes erect and dull, and after a few days it comes off, leaving a bare patch about the size of a sixpenny piece, but may extend up to 1½-2 inches in diameter, when growth ceases. The bare surface is slaty-grey in colour and is covered with epithelial scales. There is very little pruritus, and after extension of the lesion ceases the hair soon begins to grow again, and is often slightly darker in colour than the surrounding hair as in the case of trichophytosis. There is the natural tendency to spontaneous recovery unless infection is maintained by grooming utensils, harness, clothing, etc.

Contagion.—Both forms are contagious from horse to horse, to dog and cat, and to the human being—trichophytosis causing the more deeply seated and more serious lesions in man. Trichophytosis of the horse is also contagious to cattle.

Ringworm in the Ox.—Trichophytosis is the only form of ringworm met with in cattle; the commonest being *T. mentagrophytes.* It is especially common in young animals, the lesions being met with chiefly about the head, face, and lips, eyelids, and neck. It may, however, be met with in any part of the body, although the legs are very rarely affected. The lesion commences as a raised ring-like patch on which the hair is erect; then epithelial scales become heaped up to form a greyish-white or greyish-yellow crust. The lesions are usually numerous and confluent, each one extending up to about the size of a five-shilling piece. The infected area, however, may be very large owing to confluence. The crust is at first firmly adherent, and if forcibly removed leaves a bleeding surface. If left alone, the crust becomes raised by a slight purulent discharge and may be shed. This crust contains the fungus, and the hairs which may be seen in it have the characteristic frayed ends. The discharge on the bald patch dries and forms a thin scab under which healing takes place, and later hair grows again.

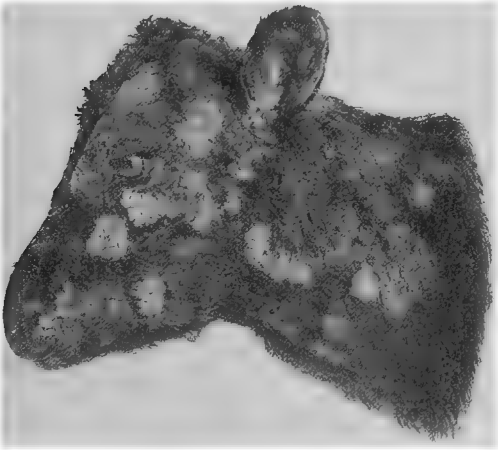


Fig. 82.—Calf with ringworm.

In adult cattle there is as a rule no systemic complication, but in badly affected young stock general unthriftiness is set up. The degree of irritation is variable, but in the majority of cases calves tend to rub themselves against gate-posts and other obstacles and thus set up centres of infection and re-infection. Ringworm in cattle may disappear spontaneously in three to four months, but through lack of attention it may assume a chronic character.

Contagion.—Ringworm in cattle may be transmitted to other cattle, to horses, to dogs, and also to the human being.

Ringworm in Sheep.—Ringworm is very rare in sheep, but trichophytosis occasionally occurs. The lesions are met with on the shoulders, neck, and chest, and may cause the fleece to be matted into tufts. The individual lesions may attain the size of a five-shilling piece but may become confluent. It is usually associated with considerable pruritus, and as the result of scratching and rubbing a ragged fleece is produced.

Ringworm in the Pig.—Ringworm in the pig is exceedingly rare, but one or two cases of trichophytosis have been observed. The lesions are quite small and crust formation is not very pronounced. There is an absence of pruritus.

Ringworm in the Dog.—Four varieties of ringworm are met with in the dog, due to the following parasites: *Trichophyton*, *Microsporon*, *Eidemella*, and *Oospora canina*; the last named causes favus in the dog. The lesions usually commence on the head (particularly the eyelids) and limbs, and may spread to other parts of the body.

The lesions other than favus correspond closely with those occurring in horses and cattle, and consist of circumscribed accumulations of crusts extending up to the size of a five-shilling piece; the hairs become frayed and loose and detached from their roots (*T. tonsurans*). There is usually some irritation, as the result of which scratching takes place, and the crusts become removed, leaving bald patches. Sometimes there are small pustules developed under the scab owing to invasion of the hair follicles.

In microsporosis the accumulation of crusts is less marked, and in many instances there is simply the loss of hair with the production of small grey scales over the patch. This form is less irritable than trichophytosis and is undoubtedly responsible for a considerable number of cases of resistant patchy scurfiness met with in the dog.

In favus ringworm due to *Oospora canina* the lesions consist of raised circular patches with yellowish-grey crusts with a cupped or honeycombed surface. The crust consists mainly of mycelial growth. Not infrequently the skin is very much thickened for some time before the crusts make their appearance, and this has given rise to the diagnosis of tumefaction of the skin.

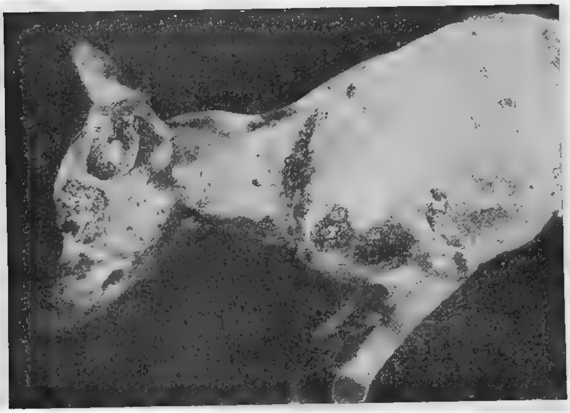


FIG. 83.—Ringworm in the dog. This animal was destroyed in a condition of extreme emaciation.

Contagion.—Each form of ringworm is transmissible from dog to dog and cat, and from dog to the human being. Trichophytosis and microsporosis are transmissible to the horse, and trichophytosis to cattle.

Ringworm in the Cat.—Three kinds of ringworm are met with in cats, viz. Trichophytosis, Microsporosis, and Favus. The two former are met with on the head and neck and chest, although they may also spread to other parts of the body. They are very similar in appearance to the lesions in the dog.

Favus ringworm is far more common and more important in the cat than the other forms, and the cat is the only species in which favus is very frequent. It is commonly due to *Achorion Quinckeanum* (mouse favus), and sometimes to *Achorion Schönleinii* (favus of man). In this form the lesions are principally met with on the paws and the head, either the forehead or the tips of the ears, but they may be met with in other parts of the body. There is usually little or no irritation. The lesions vary in size from a pin's head up to the size of a shilling. The skin is much thickened and the margins raised, while the crust is yellow and soft when fresh, but becoming greyish-yellow and powdery when old. The surface is frequently cupped. When the crust is removed the skin is usually smooth, and may either be dry or slightly moist, reddened, and somewhat inflamed. Sometimes the lesion is limited to the claws, when the characteristic cupped feature is lacking.

Favus ringworm has not the same tendency to disappear spontaneously as the so-called common ringworm (*T. tonsurans*).

Contagion.—Each form of ringworm of the cat is transmissible to animals of the same species, to the dog and to the human being—*Trichophyton* and *Microsporon* to and from the horse. Favus ringworm of the cat is most frequently contracted from mice and rats, and is also transmissible to man. On this account it has been suggested that *Achorion Schönleinii* and *A. Quinckeanum* may be one and the same parasite somewhat modified owing to long residence on different species.

Treatment of Ringworm.—Affected animals should be isolated and should be looked after by separate attendants. All litter, fodder, or other material that may have been contaminated, should be destroyed by burning, and a very thorough disinfection of the walls, partitions, and floors must be carried out. Woodwork should be either burnt or scorched by means of a plumber's lamp, and scraped and liberally treated with a strong disinfectant. If lime-wash is used it should contain carbolic acid, half a pint to the gallon, and is most effective if applied hot. In the yard or shed all posts and troughs should be disinfected, and for this purpose a coating of tar will often be found convenient and effective. Disinfection of harness, grooming utensils, rugs, and clothing must be carried out, preferably in the steam sterilizer if possible. Cats and dogs should be prevented from running into stables or cowsheds where ringworm exists, since they may be the means of continuing or disseminating infection. Vermin such as mice and rats must be exterminated. As a general preventive measure in young stock cleanliness should be observed, while the maintenance of good bodily condition by generous feeding stimulates resistance against infection. It should also be remembered that most forms of ringworm are transmissible to man, so that pet animals should be isolated when affected. Children are often infected from cats and *vice versa.*

Curative treatment in most cases is simple and effective, although in large studs it may give rise to considerable difficulty. The hair should be removed from round about the ringworms and the crusts well softened with strong alkaline solution. They should then be removed and all the debris should be carefully burnt. Avoid spreading the fungus by grooming over the part. After removal of the crusts the lesion must be dressed with a parasiticide, of which there is a considerable choice. They are best applied in the form of an ointment. Amongst the best will be found biniodide of mercury ointment 1 in 50, iodine ointment, white precipitate ointment, salicylic ointment 1 in 16, chrysarobin ointment 1 in 50. Tincture of iodine, and corrosive sublimate in spirit 1 in 200 are very effective. The last named should not be used over a very extensive area. Nitrate of silver 5 per cent freshly prepared is also good. For widespread lesions any of the following dressings will be found serviceable. Sulphur 1, pot. carb. 1, ol. picis 1, with lard or oil 8; solutions of calcium sulphurata and potassium sulphurata, solution of sulphate of iron or perchloride of iron. For very obstinate cases over a limited area, croton oil may be used, or a combination of chloral-hydrate, carbolic acid, and tincture of iodine in equal parts. Either of the last two dressings must be used with caution, and only under professional supervision. The use of X-rays has been found of very great service in the treatment of ringworm in the human being, and may be adopted in the case of dogs if the client will sanction the expense. Whatever method of treatment is adopted, it is important to realize that all diseased hair must first be shed or forcibly removed, otherwise it is impossible to kill the fungus.

Internal remedies may be given to reduce irritation, such as salines, tonics, and arsenic. A liberal diet is necessary.

It is often difficult to say when ringworm is actually cured, and it is only safe to pronounce a cure when there is no longer a scaly condition of the skin and a good smooth crop of new hair grows.

G. H. W.

MANGE MITES

Of the animal parasites which cause skin affections in the domestic animals none are of such practical importance as the mange mites. They belong to the same order (Acarina) as the ticks, and they are grouped under two families—Sarcoptidæ and Demodecidæ. They are all of small size and are best identified under a low power of the microscope.

I. Sarcoptidæ

The mange mites belonging to the Sarcoptidæ have a certain amount of resemblance to the cheese and hay mites, with which they are sometimes confused. They have all rounded, greyish bodies, convex above and flat below. There is no division of the body into thorax and abdomen. The mouth parts (comprising maxillæ, mandibles, palps) are arranged in the form of a conical rostrum placed at the anterior portion of the body. The body is covered with a tough, chitinous envelope, marked by wavy, parallel lines and carrying at places small spinules or hairs. There are four pairs of jointed limbs in the adult, the two anterior pairs being attached to the body close to the rostrum, and the two posterior pairs towards the posterior third. Each of the limbs is composed of five segments, and the last article is prolonged by an ambulacral sucker or fine hair. The parasites are not provided with tracheæ and the sexes are separate. The males are always smaller and fewer in number than the ovigerous or egg-bearing females.

The following are the genera into which this group of mange parasites is divided: *Sarcoptes*, *Notœdres*, *Cnemidocoptes*, *Psoroptes*, *Chorioptes* (or *Symbiotes*), *Otodectes*.

The distinguishing features of each of these genera may be stated as follows:

Sarcoptes.—This mite is hardly visible to the naked eye. It is oval in shape. The rostrum is stout, short, and horse-shoe shaped, and provided with two membranous cheeks. There is a Y-shaped thickening of the envelope immediately behind the rostrum.

The dorsal surface of the body presents a rough aspect from the presence of a considerable number of thorn-like spinules directed backwards. Towards the central portion of the dorsal surface the spinules are small and numerous over a considerable area. The limbs are stout, short, and thick. The anterior two pairs are placed at the side of the body close to the head, while the posterior two pairs are attached to the under aspect of the posterior third, and when the mite is examined from the dorsal aspect only the hairs which are attached to the extremities of these limbs can be seen projecting from the margin of the body. In the adult female the first and second pairs of limbs, and in the male the first, second, and fourth pairs, carry ambulacral suckers. Each sucker is small and bell-shaped, and

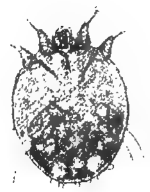


Fig. 84.—*Sarcoptes equi*. Ovigerous female.

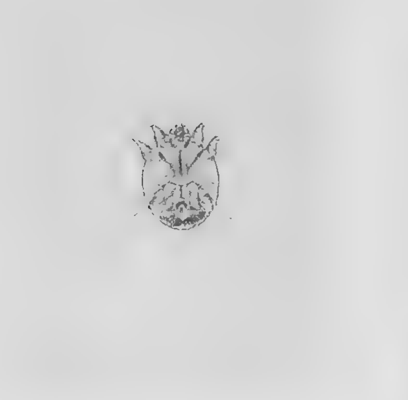


Fig. 85.—*Sarcoptes equi.* Male.

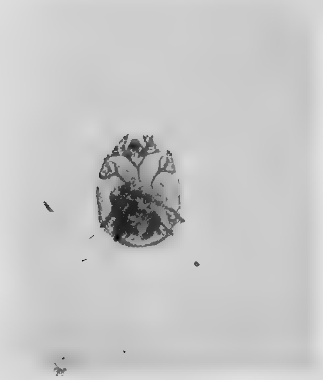


Fig. 86.—*Notœdres cati.* Female.

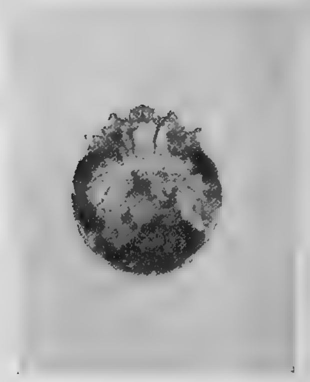


Fig. 87.—*Cnemidocoptes mutans.* Ovigerous female.

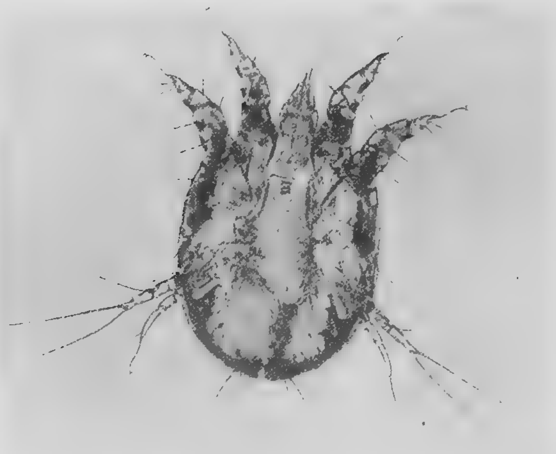


Fig. 88.—*Psoroptes equi.* Ovigerous female.

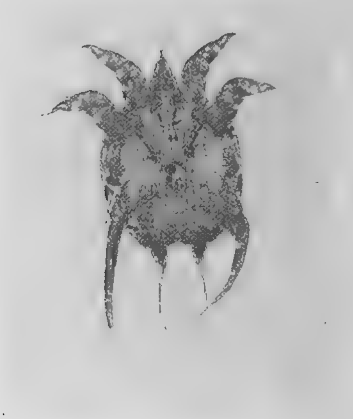


Fig. 89.—*Psoroptes equi.* Male.

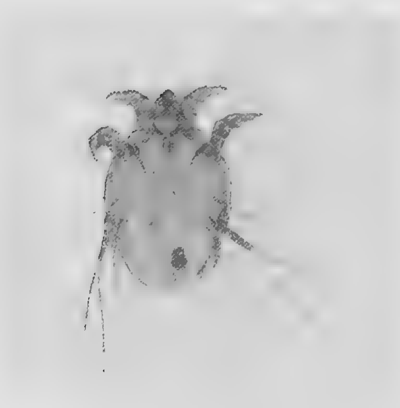


Fig. 90.—*Symbiotes equi.* Ovigerous female.

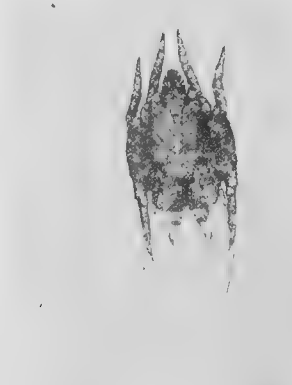


Fig. 91.—*Symbiotes equi.* Male.

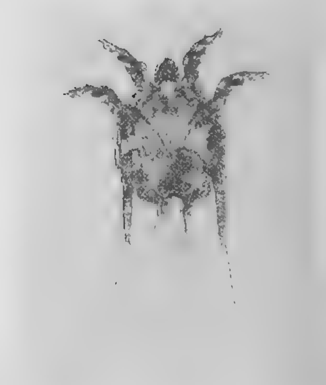


Fig. 92.—*Symbiotes equi.* Male.

attached by a fairly long, thin, unsegmented stalk to the extremity of the limb. The male has no copulatory suckers or abdominal appendages. The anus is terminal. The parasite is oviparous.

Notœdres.—This genus has only recently been separated from the sarcoptes and used to be called *Sarcoptes minor*. It is much smaller than the sarcoptes, more nearly spherical in shape, the lines on the dorsal surface of the body are arranged in concentric circles, and the anus is dorsal.

Cnemidocoptes are the sarcoptes of birds. The adult female is more spherical than the sarcoptes proper, shows no spines on the body, and the limbs are extremely short and are not provided with ambulacral suckers. The male carries suckers on all the limbs. The cnemidocoptes are ovoviviparous.

Psoroptes (or **Dermatodectes**).—The adult psoroptes are the largest of the mange mites, and can readily be seen as grey specks with the naked eye. They are oval in outline, the rostrum is long and conical, and not provided with membranous cheeks. The limbs are long and attached near the margin of the body. The first, second, and fourth pairs of limbs in the ovigerous female, and the first, second, and third in the male, terminate in ambulacral suckers. Each sucker is tulip-shaped, fluted at its free border, and attached by a long stalk divided into three segments. The third pair of limbs in the female is prolonged each by two long hairs. The anus is terminal. In the male there are two round copulatory suckers near the posterior extremity, and a round projection, the abdominal prolongation, carrying a bundle of hairs at each side of the anus. These two sets of organs facilitate coupling of the sexes.

Chorioptes or **Symbiotes** are distinguished from the psoroptes in that they are smaller, the rostrum is shorter and triangular in shape, and the ambulacral suckers are large and bell-shaped and attached by very short unsegmented stalks. The male carries suckers on all the limbs, and possesses two rectangular abdominal prolongations each carrying four bristles. The female carries ambulacral suckers on the first, second, and fourth pairs of limbs.

Otodectes.—This genus is commonly called *Symbiotes auricularum*, and used to be classified with chorioptes. Otodectes differ from the chorioptes chiefly in two details of structure—the adult female is without suckers on the last two pairs of limbs, and the abdominal prolongations are not distinct in the male.

On account of the varying importance of these genera it is often necessary to identify the particular genus under observation. Differentiation is especially important between sarcoptes, psoroptes, and chorioptes. For this purpose special attention should be paid to the appearance and disposition of the ambulacral suckers.

Varieties of these Genera.—All the domestic animals are subject to attack by a variety of one or more genera of these mange mites. The varieties of each genus are not easily distinguished from one another, but are proper to the species of host on which they are found. Each variety lives and thrives only on the species which acts as its common host. The following are the common varieties, and they are named according to the species of host on which they are found :

Man	*Sarcoptes scabiei*, var. *hominis.*
Horse	*Sarcoptes scabiei*, var. *equi.*
Ox	*Sarcoptes scabiei*, var. *bovis.*
Camel	*Sarcoptes scabiei*, var. *cameli.*
Sheep	*Sarcoptes scabiei*, var. *ovis.*
Goat	*Sarcoptes scabiei*, var. *capræ.*
Pig	*Sarcoptes scabiei*, var. *suis.*
Dog	*Sarcoptes scabiei*, var. *canis.*
Rabbit	*Sarcoptes scabiei*, var. *cuniculi.*
Ferret	*Sarcoptes scabiei*, var. *furonis.*
Cat	*Notœdres cati*, var. *cati.*
Rabbit	*Notœdres cati*, var. *cuniculi.*
Poultry	*Cnemidocoptes mutans.*
Poultry	*Cnemidocoptes lævis gallinæ.*
Pigeons	*Cnemidocoptes lævis columbæ.*
Horse	*Psoroptes communis equi.*
Ox	,, ,, *bovis.*
Sheep	,, ,, *ovis.*
Goat	,, ,, *capræ.*
Rabbit	,, ,, *cuniculi.*
Horse	*Chorioptes equi.*
Ox	,, *bovis.*
Sheep	,, *ovis.*
Goat	,, *capræ.*
Dog	*Otodectes cynotis canis.*
Cat	,, ,, *cati.*

Life-history of the Mange Mites belonging to Sarcoptidæ.—These mites are essentially permanent parasites, and their development can only be maintained on the bodies of their hosts. The stages in the life-history of all the genera are (*a*) the egg ; (*b*) the hexapod larva ; (*c*) octopod nymph ; (*d*) male and pubescent female, and (*e*) ovigerous female.

Sarcoptes.—The sarcoptes differ from the psoroptes, chorioptes, and otodectes in the burrowing propensities of the ovigerous female.

The ovigerous female sarcoptes burrows into the epidermis and there forms a small straight or curved channel, the gallery, in a few hours. It then begins to lay eggs. The female will be found at the end of the gallery with the eggs arranged in a row behind it. The rate at which the eggs are laid is difficult to make out. I have identified one case in which the eggs must have been laid at the rate of two per day. As a rule only one egg can be seen in the body of the female. In one exceptional case I noted five eggs in the body of a sarcoptes. The gallery of the parasite cannot usually be identified in the skin of the domestic animals because of the pigmentation of the skin and the

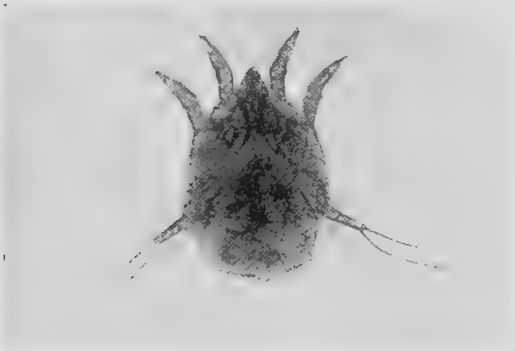


Fig. 93.—*Otodectes cynotis canis.* Ovigerous female.

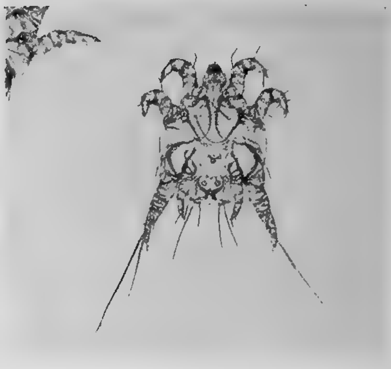


Fig. 94.—*Otodectes cynotis canis.* Male.

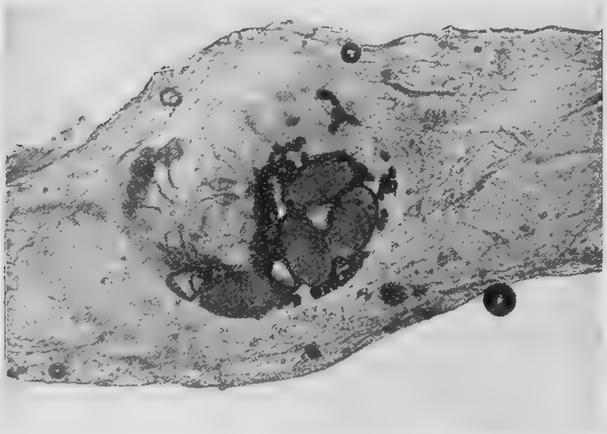


Fig. 95.—Section of skin showing gallery of *Sarcoptes scabiei* var. *canis* with ovigerous female and four eggs.

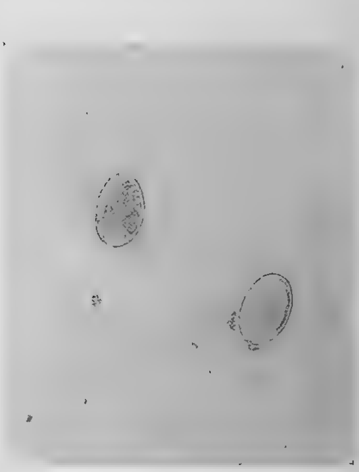


Fig. 96.—Eggs of *Sarcoptes equi.*

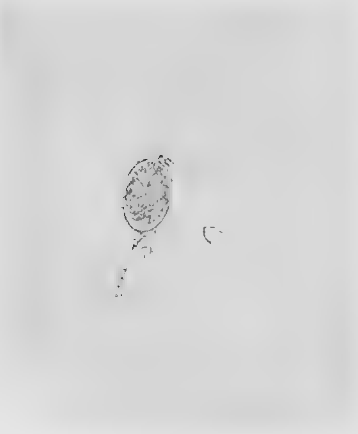


Fig. 97.—Egg of *Sarcoptes equi* containing larva.

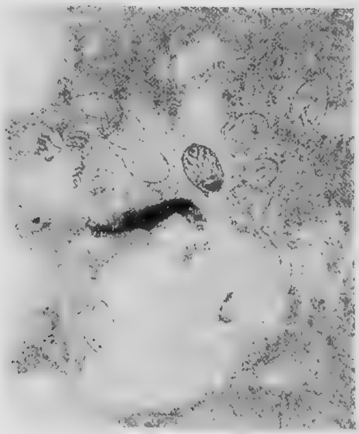


Fig. 98.—*Sarcoptes equi.* Scraping showing larva in egg and empty eggs.

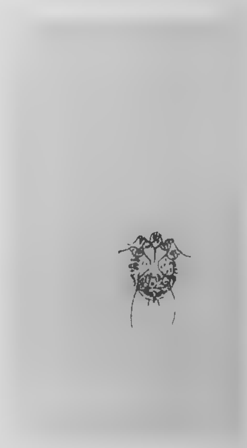


Fig. 99.—*Sarcoptes equi.* Hexapod larva.

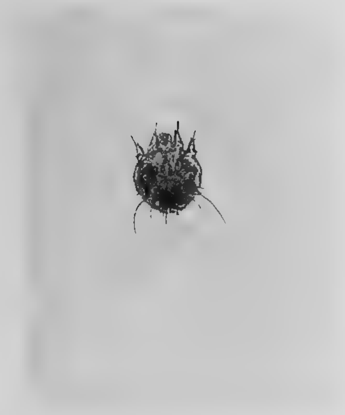


FIG. 100.—*Sarcoptes equi.* Octopod nymph.

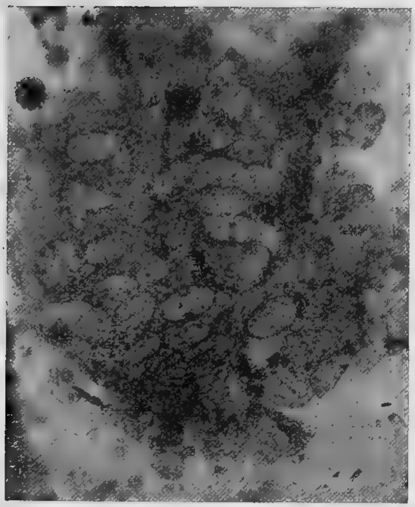


FIG. 101.—*Notœdres cati.* Nest of eggs.

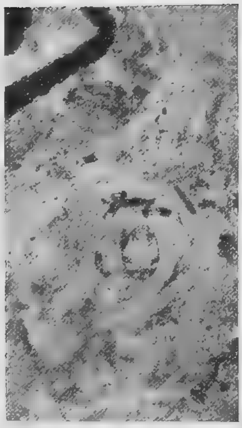


FIG. 102.—*Notœdres cati.* Egg with larva.

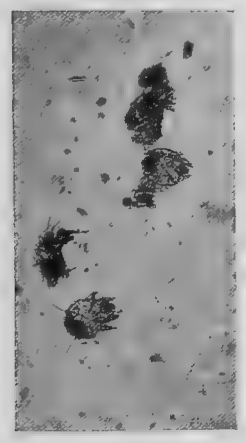


FIG. 103.—*Notœdres cati.* Larva and moults.

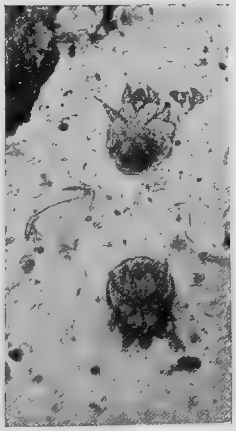


FIG. 104.—*Notœdres cati.* Male, female, and egg.

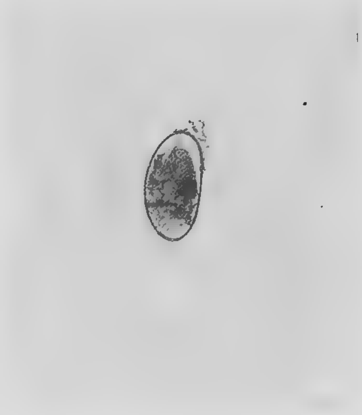


FIG. 105.—*Psoroptes equi.* Egg

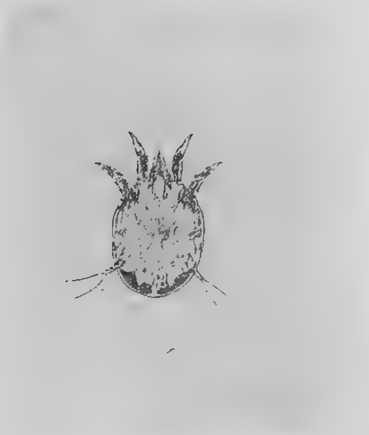


FIG. 106.—Larva of *Psoroptes equi.*

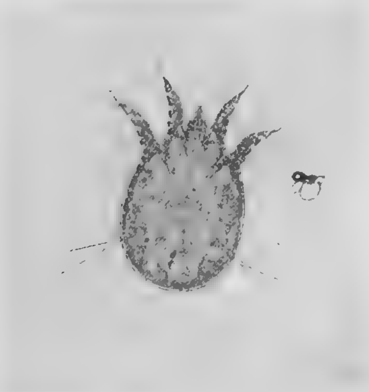


FIG. 107.—*Psoroptes equi.* Nymph.

presence of a thick coat. Occasionally in the dog and pig, where the skin is thin, the entrance to the gallery may be seen as a little dark spot on a slight elevation.

The egg is ovoid in shape, white and glistening. Its contents are granular, and it is covered with a thin, homogeneous, tough envelope. The number of eggs laid varied from 10 to 30, 15 being probably an average number. In the egg sometimes the larva may be seen in various stages of development. When fully formed it appears with its limbs under the body and is quite characteristic. After about 3 to 7 days the larva hatches out. In doing so, it splits the envelope at one end and the empty egg presents some resemblance to an oyster shell. The larva afterwards leaves the gallery, and the further stages in the development of the parasite are passed through under the scales and débris of the skin.

The *larva* is very small, with a rostrum and chitinous envelope like the adult. It is provided with three pairs of limbs (hence the term hexapod larva), the first two pairs carrying suckers, the last pair being prolonged by hairs. It gradually increases in size, and after 3 or 4 days moults. It undergoes certain changes, casts off its old envelope, and develops into the nymph.

The *nymph* is slightly larger than the larva; it is provided with four pairs of limbs similar to those of the adult female, but its sexual organs are not developed. After a few days it moults and becomes changed into the male or pubescent female. After coupling with the male, the pubescent female again moults and becomes the ovigerous female. The ovigerous female presents a transverse slit, the ovulating vulva, on the ventral aspect of the body behind the last pair of limbs, and eggs begin to form within its body.

The development of one generation of sarcoptes is thus completed in 2 or 3 weeks. The parasite cannot be kept under observation throughout its development, and therefore the period it takes is only approximately given. Gerlach, in studying the *Sarcoptes scabiei* var. *hominis*, regarded 15 days as the average time for development and the average number of progeny as 15 individuals—5 males and 10 females. He therefore estimated that from one ovigerous female 1½ millions may be produced in 90 days. This is a moderate computation, and probably makes full allowance for numbers of eggs and young parasites which fall by the way and perish.

Notœdres.—In this genus the ovigerous female forms little nests in the epidermis, straight channels vertical to the surface, in which the eggs are packed together like sardines in a box.

Psoroptes and **Chorioptes** live under the scales and débris of the epidermis. They do not form galleries. In other respects their life-history is similar to that of sarcoptes.

The usual periods commonly given for the different stages in the development of psoroptes are: The larva hatches out from the egg in 3 to 7 days, moults, and becomes changed into the nymph in 3 or 4 days. The nymph moults in 3 or 4 days and the male or pubescent female is formed. The latter after coupling with the male moults in 3 or 4 days and becomes the ovigerous female. The latter begins to lay eggs in about 4 days. The nymphal stage of

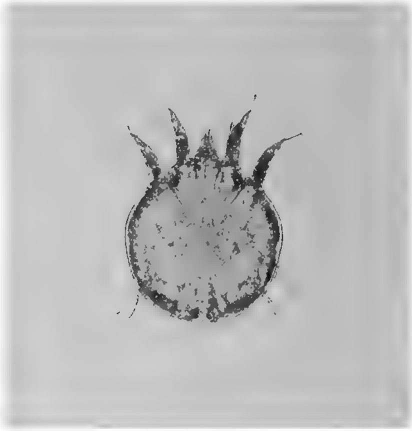


Fig. 108.—*Psoroptes equi.* Pubescent female.

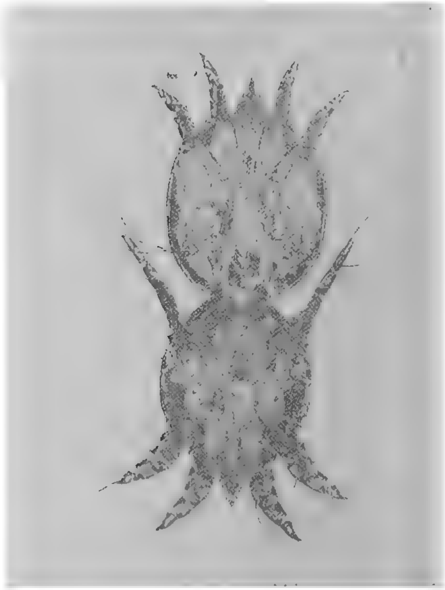


Fig. 109.—*Psoroptes equi.* Male and female *in copula.*

these genera resembles in the appearance of its limbs the ovigerous female. The pubescent female has no suckers on the last pair of limbs, which are prolonged by two fine hairs, and is provided with a little round knob on either side of the anus for attachment to the copulatory suckers of the male during coupling. Coupling has not been noted definitely in the sarcoptes, but the male and pubescent female psoroptes and chorioptes may remain attached for several hours or 1 to 2 days. The ovigerous female presents a transverse slit-like opening, the ovulating vulva, on the under aspect of the body, behind the first two pairs of limbs.

The life-history of the *Psoroptes communis* var. *ovis* has been worked out carefully by Shilston and Bedford in South Africa, and they have followed the development of the parasites throughout all its stages on the body of the sheep. The life-history as observed by them is shorter than that previously given. According to their observations, the larva hatches out from the egg in 2 or 3 days on the skin, 4 or 5 days on thick crusts, and 6 to 9 days in wool in warm weather. It moults in 2 or 3 days, the moulting process in this stage occupying 12 hours, while it lasts for 36 hours in each of the succeeding stages. The nymph which forms moults in 3 to 4 days and becomes a male or pubescent female. The latter appears before the male. Male psoroptes are in the proportion of 1 to 2, 3, or 4 females. One male may fertilize 2, 4, or 6 females. Coupling takes 1 to 2 days. The pubescent female moults in 2 to 3 days, and the ovigerous female into which it is changed begins to lay one day afterwards, or 9 days from the time of hatching of the larva.

The female may lay as many as 90 or 100 eggs, and sometimes in one day as many as 5 to 12 eggs. The egg-laying period of the ovigerous female lasts about 20 to 29 days.

Habit of Life and Intertransmissibility of Species.—As already noted, each variety of mange mite lives throughout its development and its life on the body of its host. It does not thrive as a rule on the skin of another species of animal. The psoroptes of the sheep does not live beyond the first generation on the ox, nor the psoroptes of the rabbit or goat on the sheep.

The only mange acari of the lower animals which will set up a serious skin affection in man are varieties of the *Sarcoptes scabiei* and *Notœdres*. The skin affection so set up is often temporary, but may be very extensive and require treatment before recovery is brought about. The parts usually affected are the hands, arms, and breast, but sometimes the affection extends over the body. It is quite common for the *Sarcoptes scabiei* var. *canis* and *Sarcoptes scabiei* var. *equi* to be transferred to their owners or attendants. The latter parasite has often produced serious trouble in cavalrymen and other soldiers in war time who come into intimate contact with mangy horses, their clothing, or harness.

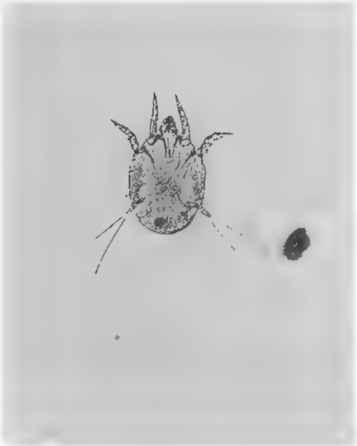


Fig. 110.—*Symbiotes equi.* Larva.

Sarcoptes scabiei var. *equi* is said also to affect cattle, *Sarcoptes scabiei* var. *ovis* to affect horses, cattle, and dogs, *Sarcoptes scabiei* var. *canis* horses and sheep; the *Notœdres cati* is sometimes transferred to dogs, and is said also to produce mange in horses and cattle.

On its proper host the mange mite lives on the juices, excretions, and débris of the skin. It penetrates the epidermis with its strong rostrum and sucks up the juices which exude.

The *length of life of the parasites on the host* is not definitely known save for a few varieties. Female sarcoptes are said to die in 3 to 5 weeks after laying eggs; the males live for about 6 weeks. The life of the ovigerous female psoroptes of the sheep is 30 to 40 days including the developmental stages. The male lives for 22 to 34 days after moulting from the nymph. The mortality of the parasites is affected by various circumstances connected with the condition of the animal and the excretions of the skin.

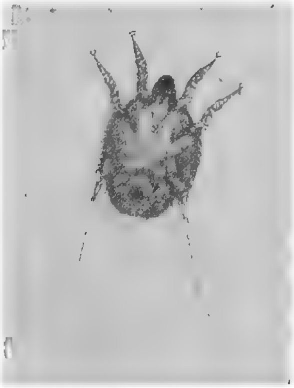


Fig. 111.—*Symbiotes equi.* Nymph.

Off the host the parasites live only for a short time, in most cases less than one month. The sarcoptes die most rapidly; it is seldom they can be kept alive for more than a few days. In a dry atmosphere they perish in 5 or 6 days. When kept moist they do not live more than 14 days. Gerlach noted feeble signs of life in a sarcopt of the horse kept moist on a fragment of skin on the 24th day, but death had occurred by the 28th day. Psoroptes live apart from their host as a rule not more than 20 days. I have seen them live for 24 days, and the ultimate period observed by Stockman was 30 days. In dry air they are dead by about the 10th day. Chorioptes may live a little longer than the previous genera. Delafond and Bourguignon kept them alive from 60 to 65 days in boxes placed in a stable, but their observations have not been confirmed.

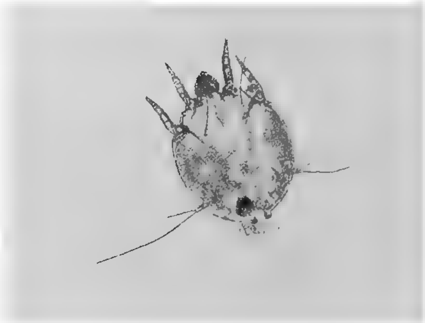


FIG. 112.—*Symbiotes equi.* Pubescent female.

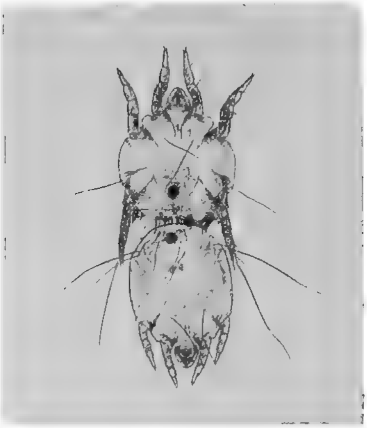


FIG. 113.—*Symbiotes equi.* Male and female *in copula.*

It will be noted in observations made with the object of ascertaining the length of life of these acari apart from the host, that the young parasites die first and the adult ovigerous females survive longest. After a few days, 10 days in the case of the psoropt, the chief evidence of life shown consists in slight movements of the limbs. It is probable, therefore, that when the parasite becomes enfeebled, it will not thrive or propagate when placed on its proper host. The eggs retain their vitality at most for about 10 days, and then may be hatched out either in an incubator or on the body of the animal which it usually attacks. Gerlach, however, claims to have seen the eggs of the sarcopt of the horse hatch out after four weeks.

The Effects of the Mange Mite.—The mange parasite produces its effect by puncturing the epidermis with its rostrum and injecting certain irritating secretions. Gerlach showed that this was the chief means by which the parasite sets up skin disease. He punctured the epidermis with the point of a needle, withdrew the latter and charged it with the juice of a crushed parasite. He then introduced the needle into the tract he had made in the epidermis. When the point of the needle reached the dermis an itching, painful sensation was produced, followed by the production of a papule and vesicle. The effect was more marked in the case of the psoroptes than with the sarcoptes, and was least severe with the chorioptes. The movements of the acarus on the skin cause little trouble. The ovigerous female sarcoptes also does some damage by burrowing into the epidermis.

The disease of the skin which is set up is referred to as *scabies, mange, itch, scab,* or *acariasis.*

The skin affection produced is of the nature of a dermatitis, and is accompanied by itching or pruritus. The pruritus is more marked when the animal is heated as the result of exercise, or when kept in a warm house, and is not so evident when it is exposed to cold weather. It varies in degree with individuals, is perhaps greatest in the finer breeds and in adults, and is in no way related to the number of parasites present. Some of the most itchy patients may present only a few parasites on examination of scrapings, whereas in others where pruritus is not very apparent the acari may be in abundance on the skin. It is often greater with the psoroptes than sarcoptes, and least evident with chorioptes. On account of the pruritus the animal rubs, scratches, and tries to bite the affected areas, so that at the points which it can reach, the hair is rubbed off in patches and abrasions and sores are produced. These obscure the lesions proper to the disease.

The dermatitis follows on the puncture of the epidermis by the rostrum of the parasite. A small papule appears, no larger than a pinpoint or pin-head, and is at first reddened. A little serous fluid may exude, which coagulates on the surface and may agglutinate a small tuft of hairs. This tuft is afterwards cast off. The epidermis proliferates, the skin becomes thickened, scaly and dry, and covered with crusts. From repeated and simultaneous punctures of several parasites the areas extend. The gallery of the sarcoptes appears as a red point on a small elevation, but it is seldom seen, and therefore the lesion formed by this mite is difficult to distinguish from that produced by the other mange acari save by reference to the site of the lesion or the identification of the parasites. The nodular condition of the skin in the first stage is sometimes the only thing noted, the reddening seldom on account of the pigmentation of the skin, and so the lesion often presents no very characteristic feature.

At some places in the later stages the skin becomes wrinkled, especially over areas where the subcutaneous tissue is loose. The loss of hair is very extensive. Vesicles are seldom, if ever, seen, and pustules only occur from the inoculation of pyogenic organisms.

As the result of the restlessness and the absorption of toxic agents injected by the parasite, the affected animal may lose condition and even become emaciated. This is more noticeable in attacks of sarcoptes and psoroptes, and is most likely to occur when the skin receives no attention, when the animals are left in a dirty condition, are poorly fed and overworked.

Transmission of the Parasite.—In all cases the affected animal is the chief source of infection. The danger of transmission is in direct proportion to the number of parasites and the extent of the disease. When the parasites are few in number and the coat of the animal is unbroken, the acari are not readily transferred to the neighbouring animals.

Transmission rapidly takes place where animals are closely associated. The rapid spread of the disease is often associated with dirt and neglect, when the animals are neither groomed nor washed at frequent intervals; when they are in poor condition from overwork, exposure, or under-feeding. The parasites grow and thrive best when they are not disturbed.

The parasites may be transferred by direct contact or through intermediate carriers, such as common rubbing-posts, grooming utensils, clothing, harness, by the hands or clothes of attendants, or by the litter. Susceptible animals may become infected by introduction to houses, trucks or other vehicles, yards or fairs recently occupied by animals affected with mange.

It is curious that outbreaks of mange as a rule are most numerous in the domestic animals in the winter time. The explanation of this occurrence is not very clear. It may be related sometimes to the length of the coat at that time and the consequent lack of evidence of the disease until it is far advanced, perhaps also to atmospheric conditions. It cannot be connected with the temperature at which the acari are kept on the skin. Certainly when off the body of their host the parasites are quite dormant in the cold, whereas at body temperature they are very active and move about very rapidly. I have noted the sarcoptes moving at the rate of 1½ inches per minute on the surface of the skin. It is also interesting to note that as a rule chorioptes are not so rapidly transferred to in-contact animals as the other genera of mange mites belonging to the Sarcoptidæ.

The interval which elapses after infection until appreciable symptoms appear is rather indefinite, and varies to some extent with the number of parasites which pass on to the host. In an average case the symptoms will be noted within a month. If, however, only a very few parasites are transferred, or if the animal is well fed, well kept, and frequently groomed and washed, the disease may not be apparent for two, three, or four months. This may account for the cases which appear without any distinct history of contagium. In some of such cases it has been thought that certain places have remained infected for years, and that these places are the source of the infection, but from our knowledge of the life-history of the parasites that would appear to be an erroneous opinion. The application of acaricide dressings will also delay the progress of the disease, and certain conditions of the skin may militate against the rapid propagation and increase in numbers of the acari. That has been noted in sheep which have an excessive amount of yolk in the wool.

Diagnosis.—The diagnosis of each form of mange is made from the appearance and position of the skin lesion, the itchy condition, and the identification of the parasite causing the lesions.

A clinical test commonly applied is to scratch the part of the skin showing evidence of disease. If the animal stretches out or turns round its head and makes nibbling movements of the lips, the condition is often diagnosed as mange. In the case of the dog or cat the animal makes scratching movements with the hind limb when the affected part is rubbed. The evidence obtained in this way of the presence or absence of mange is not absolutely conclusive; it is only of relative importance, since in affections apart from mange itchiness or pruritus is often noted. The diagnosis is made absolutely certain only when the parasite is discovered, and a search should, as far as possible, always be made to ascertain the cause of a suspicious skin disease.

This may be done in one of several ways.

(1) The surface of the affected area of skin may be examined with a hand lens. Where psoroptes or chorioptes are the cause of the skin affections, sometimes they may be seen

as little grey moving specks on the skin or the hairs. These specks can be picked off with a mounting needle or on the point of a knife, deposited on a glass slide, mounted in a little glycerin or Farrant's medium, and the particular genus to which the parasite belongs identified under the low power of the microscope. A magnification of 20 to 50 only is required, and in the inspection of the acarus a dull light is best for the discovery of its characteristic features.

(2) In most cases scrapings of the skin have to be taken and examined before the parasite can be found. This is especially the case with sarcoptes. In order to avoid difficulties in finding the parasite great care must be taken in selecting the area from which the scrapings are to be removed. Dark scabs covering sores and the outer portions of hair or wool fibres are useless. The scrapings should be taken from recent lesions where the skin is lumpy or nodular or covered with crusts easily broken down. In sheep scab the best material to examine is the yellow waxy matter at the base of the wool fibres in the more recent lesions.

In parts of the skin which have been dressed with a parasiticide preparation it is often difficult or impossible to discover the acari. In taking the scraping from the area, the hair, if long, should be clipped off. If there is a wind blowing at the time, the part should be moistened in order to prevent the scrapings being blown away. The skin over an area of 1 or 2 inches is scraped with a moderately sharp knife until serous fluid or blood exudes from the surface. This is done in all cases in order to ensure that sarcoptes which burrow into the epidermis, if present, will be included. The scrapings are then placed in a small box or petri dish, and put in a moderately warm place, as an incubator, for half an hour or more. The acari will often creep out from among the scales, and may be identified as in the first method. Obviously this method will not be successful when eggs only are present, but it is often useful.

(3) A portion of the scrapings is mixed in a watch-glass or a slide with a 10 to 40 per cent solution of caustic potash or soda. This is done to clarify the scales and decolorize the melanin present. This is effected after a few minutes in some cases, but often takes half an hour or more. The solution has no effect upon the parasites to interfere with their identification. The mixture is then spread out in a thin layer on a clean glass slide, and a second slide squeezed down over it to ensure that the preparation is uniformly thin and transparent and easily inspected. The preparation is then examined under a low power of the microscope with a moderate light. Any or all of the stages in the development of the acari may be detected—eggs, larvæ, or adults. Their discovery may be made after a few seconds or minutes, but in some cases only after several preparations have been made and examined. This method is quite satisfactory, and can be relied upon to give good results in most cases.

(4) In order to hasten the action of the caustic alkali solution upon the scrapings, a fourth method has been introduced. The scrapings are put into a test-tube and shaken upon with a 10 per cent solution of caustic potash or soda. The contents of the tube are then boiled for about five minutes over a Bunsen burner or a spirit lamp. The skin débris which remains, portions of the hairs, and the parasites are quite transparent. The mixture is then centrifugalized for a few seconds and the solid particles are deposited at the bottom of the tube. The supernatant fluid is canted off, a little glycerin is added and mixed up with the deposit. A smear of this material is made on a slide and examined under a low power of the microscope. The parasites are found very readily if they are present, even in moderate numbers, in the skin scrapings. This method is particularly useful when the parasites are few and a large number of specimens have to be examined.

It has to be remembered that in the examination of scrapings hay mites and other acari may be found which have nothing to do with mange. These hay mites have as a rule a smooth envelope, and their limbs end in hooklets.

Treatment.—The general lines of treatment are the same for all cases of mange. To be successful the treatment must be thorough, and that is the chief secret of success. Steps are taken to facilitate the action of some parasiticide dressing to be applied to the skin. The hair is clipped off the whole of the body in order to make sure that no spot escapes treatment when the body is affected. The hair is then burnt. The skin is afterwards washed or scraped to get rid of the scales and débris of the skin and permit of the penetration of the dressing and its action upon all the parasites present. When the skin is washed with soap, the whole of the soap is thoroughly rinsed off with water and the skin dried.

The dressing to be applied has for its object the destruction of the mange parasites. Many drugs are efficient in this respect, but those used must not only be efficient parasiticides, but in the proportion used must have no irritating effect upon the skin. Some dressings are discarded on that account. They may produce a more serious dermatitis than mange itself. The active agents in the dressings commonly used are sulphur and its compounds, coal-tar derivatives, and mercurial compounds. Arsenical preparations are used commonly in sheep, and sometimes in the ox. Tobacco decoctions are also employed, but great care must be taken on account of the danger of absorption of the nicotine and consequent poisoning. On that account also mercurial dressings must not be employed in the ox, or carbolic applications in the dog. The excipients used include water, oils and fats, such as rape oil, sperm oil, lard, train oil, fish oil. Linseed oil cannot be used, because it dries quickly and prevents the normal excretions of the skin. Some oily or fatty excipients sometimes, as in the case of rape oil, have an irritating action on the skin, probably on account of some adulterant. In mange affecting the body the applications must be thoroughly rubbed in over the whole of the skin. Where the dressing contains oil or fat, only one-half or one-third of the body should be dressed on successive days, in order to avoid chills. It is necessary to repeat the dressings once or twice a week until a cure is effected, because some parasites may escape the action of the parasiticidal agent for a time. In sarcoptic mange especially the skin should be washed every week. The animal must be fed well during the treatment. That is most important. Occasional cases recover as the result of a generous diet.

The bedding should be completely changed weekly, and the house disinfected as well as the clothing and utensils or other agents coming in contact with the patient.

A cure is probable when the skin becomes normal, the itching has ceased, and the hair is beginning to grow. As a rule sarcoptic mange takes a longer time to cure than other forms of mange. Animals affected with mange should not be allowed in contact with their fellows for a period of two or three months after treatment. Cases of relapse may occur because some of the eggs or adult parasites have survived the effects of treatment.

Prevention.—All cases of mange must be isolated, and the houses in which they have been living, the clothing, grooming utensils, harness, and any other object with which they have been in contact must be so treated as to destroy any stray acari. The latter are destroyed by heat, such as by hot water or flame of a painter's or brazier's lamp (this is useful for the floors, walls, and ironwork in a building), or by any of the ordinary disinfectant fluids. In-contact animals should also be dressed, and newly purchased animals should not be allowed to mix with the others until they are carefully examined or have been quarantined for a few weeks.

II. Demodecidæ

is the second family of acariens which contains mange acari. It includes only one genus, *Demodex folliculorum.*

This acarus is very small, worm-like, and transparent. The rostrum is prominent in front, a little narrower than the thorax, and is formed of similar parts to those noted among the Sarcoptidæ. The cephalo-thorax is separate from the abdomen, convex above, flat on the lower surface, and in the adult carries four pairs of legs which appear like little knobs at the margins of the lower surface. Each limb has three segments. The abdomen is long and somewhat conical, finely striated transversely. The anus is placed near the anterior portion of the abdomen. The demodex is oviparous.

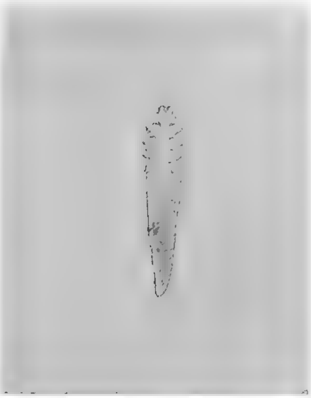


FIG. 114.—*Demodex folliculorum* var. *canis.*

Of the *Demodex folliculorum* there are several varieties, each having a separate host. They are found in man, dog, ox, pig, goat, rarely the horse and other mammals. Each variety is named after the host which it infests. Thus the variety in the dog is named the *Demodex folliculorum* var. *canis.*

The stages in the life-history of the parasite are similar to those of the other mange acari. The egg is elliptical, the larva small with three pairs of legs, and in the nymph there are four pairs of limbs with the cephalo-thorax not well separated off from the abdomen.

Habit of Life.—The demodex is found in the hair follicles and sebaceous glands of the skin, sometimes in great numbers from twenty to several hundreds.

In man it is regarded as a more or less harmless parasite, and is most frequently noted in the Meibomian glands and comedos, but sometimes it has been associated with acne.

In the dog, and occasionally in other domestic animals, it sets up one of the most serious and obstinate skin diseases. The disease is referred to as follicular or demodectic mange or acariasis. In the dog, and probably in other animals, the demodex is associated in its action with a pyogenic organism such as *Staphylococcus pyogenes albus.* This organism is probably carried by the demodex into the hair follicles and sebaceous glands. It is thus allowed to enter the tissues and set up a dermatitis, sometimes with pustule or abscess formation.

Transmission.—In the dog it appears to be only very slightly contagious. It is observed often in one or two among a litter of puppies, although they have all been in contact for a considerable time. In occasional instances among other animals a number of cases have been discovered in a herd of cattle. Possibly there is some predisposition towards the disease required for its development.

Symptoms.—There is very little pruritus in follicular mange. In some cases the skin in parts is roughened, scaly, becomes denuded of hair, reddened or pigmented, and may give off a peculiar odour. In other cases little nodules or pustules form in connection with the hair follicles. These pustules contain a thick semi-solid pus, or, when they become large, a blood-stained purulent fluid.

Diagnosis is made in the first cases, the squamous form, by examination of scrapings from the skin as in other forms of mange. In the second, the pustular form, the demodex is found in abundance in the pus from the small pustules. J. F. C.

Mange. Acariasis

The *forms of mange* are named according to the genus of acarus which causes them. Thus mange is said to be sarcoptic when due to *Sarcoptes scabiei.* The forms of mange met with in the various species of domestic animals are the following :

Horse, ass, mule	Sarcoptic, Psoroptic, Chorioptic ; rarely Follicular.
Ox	Sarcoptic, Psoroptic, Chorioptic ; rarely Follicular.
Sheep	Sarcoptic (rare) ; Psoroptic (most common) ; Chorioptic.
Goat	Sarcoptic, Psoroptic (otacariasis) ; Chorioptic ; rarely Follicular.
Pig	Sarcoptic ; Follicular.
Camel	Sarcoptic.
Dog	Sarcoptic, Follicular ; Otodectic (otacariasis).
Cat	Notœdric ; Otodectic (otacariasis).
Fowl	Cnemidocoptic (or Sarcoptic).

Mange in Equines

(*a*) **Sarcoptic mange in equines,** the most serious form of scabies in these animals, is due to *Sarcoptes scabiei* var. *equi* (Linné, 1758). This parasite was discovered and described first by Delafond and Gerlach in 1856, and has been studied in more recent years by various investigators.

The ovigerous female is about 420 μ long by 300 μ broad. The male is 250 μ long by 170 μ broad.

The disease is common in countries where there is much trafficking in horses, and its rapid spread is frequently associated with dirt and neglect, and with indiscriminate mixing of equines. It is one of the most common and serious contagious diseases which affect military horses in times of war. On account of the conditions under which the animals are kept when the disease is introduced into an army, it spreads rapidly and is extremely difficult to eradicate. The close contact of equines, hard work, dirty surroundings, want of grooming, poor feeding, and other adverse conditions, hasten the course and development of the disease. It was rampant in army horses during older campaigns, *e.g.* in the Crimea and in the South African war, and has been a source of serious trouble during the recent world war. The disease is no doubt met with in the army in peace time, but it is also brought in by remounts, and it is contracted by introduction to infected stables, railway trucks, yards and other places, and by direct or indirect contact with the horses of other armies and affected civilian horses. It is spread by direct contact, also by grooming utensils, clothing, harness, by the hands and clothes of attendants, and through infected stables or lines. The disease is not so common among thoroughbreds, because of the care and attention which they receive, and their more or less complete isolation from other breeds of equines. Carefully tended, well-fed animals are not so liable to contract the disease, and in them the disease does not develop so rapidly or extensively. Outbreaks are more frequently met with in winter.

Symptoms.—The first symptoms that may be noticed are those of pruritus. When the animal is heated, after exercise, or in a warm stable, or when well clothed, it rubs itself against posts or walls and attempts to bite the affected areas. When the affected part is scratched or groomed, the horse leans over towards the hand or brush and stretches out or turns round the head and makes nibbling movements with the lips. Well-bred animals often become frantic with the itching. The degree of pruritus varies very considerably. In horses which are kept in the open the

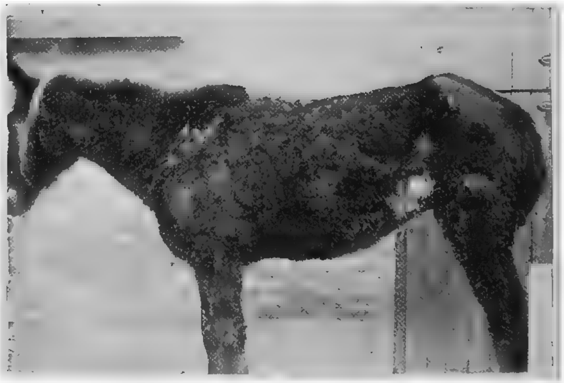


FIG. 115.—Horse affected with sarcoptic mange.

pruritus is not very evident in cold weather, and the long coat of the animal may prevent the condition from being noticed until the disease is very far advanced or the long coat is clipped off. The lesions may appear on any part of the body, but most frequently develop first on the withers, back, shoulders, neck, and sides. Thence they extend to all portions of the body, including the head, submaxillary space, under-aspect of the neck and inner sides of the thighs and forearms. The extremities of the limbs are affected only in advanced cases. The lesions appear first as little nodules on the skin, with agglutination of a few hairs over the nodules. There may be a slight oozing of yellow serous fluid which coagulates on the surface. The hairs are cast off and a dry, scaly, hairless spot is left. A number of these hairless spots coalesce and form dry, irregular, rough, scaly patches. The coat becomes dry and dirty-looking.

In neglected cases the disease frequently extends very rapidly, and large areas become involved. Only a few hairs may remain scattered over these areas. The skin becomes thickened and wrinkled in the old-standing cases, and the wrinkling is especially marked when the subcutaneous tissue is loose, such as over the withers and neck and in front of the fold of the thighs. Sometimes small pustules appear.

On account of the rubbing and biting induced by the pruritus, the skin is denuded of hair at various points, and abrasions, sores, and ulcers covered with black scabs result. This complicates and obscures the ordinary lesions.

The constant restlessness caused by the pruritus and the absorption of the excretions of the parasite bring about loss of condition and even extreme emaciation and debility. Indeed in advanced cases severe losses may be caused by this disease.

(*b*) **Psoroptic mange** is caused by *Psoroptes communis equi* (Hering, 1838). The parasite of this disease was first observed by Souting, and illustrated later in 1878 by Gothier.

The ovigerous female is about 710 μ long by 450 μ broad. The male is about 500 μ long by 330 μ broad. The disease is very highly contagious, and is transmitted in the same way and occurs under the same conditions as sarcoptic mange. It is not, however, so difficult to cure, but sometimes the two diseases are associated in the one animal.

Henry noted psoroptes on the inner side of the ears of 71 per cent of the carcases of horses from one veterinary hospital in France, where only 7 or 8 per cent were affected with psoroptic mange. 42 per cent in another hospital were also affected, although mange of any kind was rare among the equines treated at that place. He concluded that the inner side of the ear was the normal habitat of the psoroptes, and that only on occasion did these parasites leave the ear for the body and set up the symptoms of body mange.

Symptoms.—The symptoms of psoroptic mange are very similar to those of sarcoptic mange, but the degree of itching may be even more marked. The lesions appear first usually at the base of the mane, on the poll and the root of the tail, and thence extend over the body. Frequently a yellow serous coagulated material may be noted among the scales and scabs. The lesions often appear more circumscribed and moister than in sarcoptic mange, the scales thicker and more agglutinated together, and the disease does not spread over the body so rapidly.

These differences are so slight that they cannot be relied upon to differentiate the two conditions. The chief method of distinction consists in the identification of the parasite. In psoroptic mange the acarus may be seen sometimes as a grey speck moving over the surface of the lesions.

When the psoroptes are confined to the ears, slight signs of itchiness in these regions may be noted. The animal frequently shakes the head or rubs the base of the concha; occasionally it rubs the ear with the hind foot. Dust or mud may then be left on the ear with the hoof. The bottom of the auditory meatus is filled with a plug of a greyish colour sometimes an inch long.

(*c*) **Chorioptic** or **Symbiotic mange** or leg mange is due to *Chorioptes* (*symbiotes*) *equi* (Hering, 1845).

The ovigerous female is 420 μ long by 260 μ broad; the male is 305 μ long by 225 μ broad.

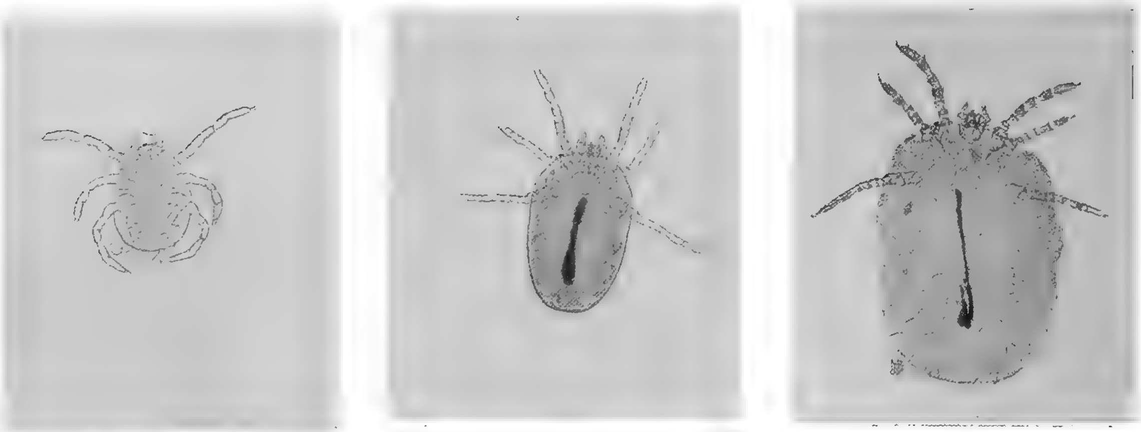
This disease is most commonly noted among heavy horses with long coarse hairs on the limbs. It is not so contagious or so serious as the other common forms, and may affect only one or two animals in a stud. Sometimes it involves a large proportion of the horses in the place. The chorioptes has been credited with producing grease; it certainly has been found in cases of grease in the horse.

Symptoms.—The disease is chiefly confined to the limbs, and the hind limbs are most commonly involved. When the horse is heated, more especially when in the warm stable, it often stamps and rubs or even bites the limb. This is the cause of night kicking in horses. Such horses may be difficult to shoe, and are more difficult to treat in foot troubles. The lesion begins first about the pastern, the skin becomes scaly and thickened, and the hair is cast off or rubbed off in patches. The disease extends upwards to the hocks or knees, and even as far as the stifles. Rarely it extends to the abdomen. In advanced cases the skin is very much thickened and scaly, and arranged in thick transverse folds covered only sparsely with hairs. The condition of the horse is not, however, much interfered with. The position of the lesions is characteristic, and the chorioptes are easily found in the hairs and scrapings. The lesions are most marked in the winter time; the animal appears to recover to some extent in the summer.

Diagnosis.—These three forms of mange are distinguished by the itchiness of the skin, the position and appearance of the lesions, and also by the discovery of the parasites in the lesions or in scrapings. The method of discovering the mange mites has already been described (p. 455).

Differential Diagnosis.—Itchy conditions of the skin are sometimes set up by other animal agencies—hay mites, lice, *Dermanyssus avium*, harvest bug; but one has to judge whether these when found on the skin are sufficient to account for the lesions present; they may complicate mange. Hay mites (various species of *Tyroglyphus*, *Tarsonemus*) sometimes cause a temporary itchiness without any very manifest lesions. With lice the itchiness may be marked, and the scurfiness and other abnormalities of the skin present are usually in direct proportion to the number of parasites. The eggs and adults (*trichodectes* and *hœmatopinus*) may easily be identified on inspection of the skin. *Dermanyssus avium* is derived from fowls or other birds which live in the lofts of the stable above the horses, and may perch on their back. The irritation they produce is very severe. Equines are attacked by the harvest bug in the autumn on certain pastures. The limbs and under-aspect of the body are affected. The parasites can easily be found and distinguished. Tick larvæ sometimes attack horses at grass. The lesions appear suddenly and consist of thick yellow scabs which agglutinate the hairs. They may affect a large portion of the body, and are not accompanied by much itching. In mane and tail mange where elf-locks and rat-tail appear, the disease is confined more or less to the base of the mane and the tail, and tends to become chronic. In chronic eczema also the lesions tend to be localized and itching is not a marked symptom, but in doubtful cases it is necessary to resort to a microscopic examination of scrapings from the lesions.

Treatment of Sarcoptic and Psoroptic Mange in Equines.—In the British Isles sarcoptic and psoroptic mange of equines are scheduled diseases dealt with under the Diseases of Animals Acts. Outbreaks of these diseases must be notified by the owner or attendant and the veterinary surgeon called in for professional advice or treatment.



FIGS. 116, 117, 118.—Harvest bug (*Septus autumnalis*) in various stages of repletion.

The animals must be isolated, placed in a separate box or stable, provided with separate utensils and, if practicable, separate attendants. The stalls from which they are removed and structures with which they have been in contact must be properly disinfected as hereafter described, and the bedding burnt.

The whole of the coat, including the hair of the mane and tail, must be clipped off. This is done in the first instance to find out the extent of the lesions, and in the second place to facilitate the penetration of the parasiticide dressing. Then the animal is washed all over with soap and water to remove the dirt, the scales and crusts, and the soap is afterwards rinsed off. There is no special kind of soap necessary. After washing, the skin is thoroughly dried and a dressing applied. Many kinds of parasiticide dressings are used, all more or less satisfactory in their way, but the chief thing necessary is to ensure that whatever one is used it is thoroughly applied. No irritant dressing should be used.

Sulphur or sulphur compounds form the base of a large proportion of mange dressings. A common and efficient application is composed of:

Sulphur, 2 parts.
Oil of tar, 1 part.
Pot. bicarb., 1 part.
Rape oil, train oil, fish oil, or lard, 8 parts.

The chief trouble here is the excipient. Rape oil sometimes blisters the skin, probably because it is rancid or contains some irritating adulterant. Clarified horse fat is now used extensively among military horses as an excipient for mange dressings and has proved eminently satisfactory. This dressing must be thoroughly rubbed into the skin all over the body from the tip of the nose to the tip of the tail, not forgetting the under-aspect of the abdomen, the inner side of the thighs, the head and the limbs. Where the lesions are strictly limited, a dressing over that part of the body and over a large area around the lesions may be efficacious, but where the disease is at all extensive the whole of the body must be treated. In cold weather, where the whole body is to be treated, a third or one-half is dressed on successive days in order to avoid chills. Exercise is useful where it is permitted. As to rugs for the animals after dressings, they are not used for military horses, but their use may be permitted in cold weather provided they are frequently disinfected. The dressing should be applied twice a week. At the end of each week the skin should be washed and a day's interval allowed before dressing again. The skin remains scaly for a time, but the itching soon ceases. The treatment must be continued until the hair begins to grow. The new hair often grows rapidly, and is darker in colour than that of the old coat.

A cure is much more rapidly effected in psoroptic than sarcoptic mange. In the former recovery may take place in eighteen to twenty days, while in the latter at least a month will elapse, and frequently a longer time, even several months in extensive cases, before recovery is complete. This difference is due to the burrowing propensities of the sarcoptes and the consequent greater difficulty of bringing the dressing into contact with these parasites.

On account of the discovery of the psoroptes on the inner side of the ears by Henry, it is advisable in psoroptic mange to apply the dressing to the inner side of the ear. M. Henry advises, as the best acaricide, cresyl in 2 to 3 per cent warm watery emulsion; 50 c.c. of this emulsion is poured into the ear two or three times at intervals of eight days.

During the period of treatment the animal must be fed well. This is an essential part of the treatment and favours recovery. Internal treatment is not necessary. Arsenic in the form of liquor arsenicalis may be given internally in advanced cases. Bedding if used should be removed every week and burnt, and the floor and walls of the box flamed with a brazier's lamp or sprinkled with a reliable disinfectant, such as 1 or 2 per cent creolin. Halters and head stall or collar should also be frequently disinfected. After recovery of the animal, the box, clothing, harness, including collar or head stall, the grooming and other utensils must be disinfected.

From the box or stall the litter is cleaned out and burnt. The walls and floors are then flamed with a brazier's lamp or sprinkled with a strong disinfectant fluid, such as 2 per cent creolin or 5 per cent carbolic acid, or douched with boiling water. Afterwards the walls and posts are whitewashed or tarred. The utensils should also be disinfected by dipping in boiling water or immersing in a disinfectant solution. Paraffin or paraffin emulsion such as that recommended by Maj.-Gen. Blenkinsop may be used for the brushes, curry-combs, clipping machine, collar, or head stall. The padding is removed from the harness and burnt, and the saddlery smeared with a disinfectant solution. The clothing, such as rugs, should be disinfected by dry heat or by immersing in 1 per cent creolin or other reliable disinfectant.

The horses after apparent recovery should not be allowed to come in contact with healthy animals for a period of three months. Relapses sometimes occur. A few parasites may survive the treatment; they do not multiply in sufficient numbers for a long time, it may be two or three months, to produce appreciable lesions.

Other dressings used for mange include:

℞ Ol. Picis, Sulphur, āā 1 part; Sapo moll., Spts. vini meth. āā 2 parts.
℞ Ol. Picis, Sapo moll āā. 1 part, Spts. vini meth. 8 parts.
℞ Cresol, Sapo moll. āā 1 part, Spts. vini meth. 5 to 10 parts.

Champetier's dressing consists of:

Sodæ bicarb.	30 parts.
Petrol	300 ,,
Oil of cade	20 ,,
Pea-nut oil	100 ,,
Hydrarg. perchlor.	1 part.
Water	1000 parts.

After applying the dressing, the horse is powdered with sulphur. This dressing is applied two or three times only. Good results are claimed by Champetier for this method.

Major-General Blenkinsop recommends the following method of treatment: After clipping, washing, and drying the horse, apply dressing of sulphur 1 part, lard, train oil, or fish oil 4 parts. Lunge the horse daily and rub the dressing in well with the hand after exercise. On the sixth day apply the following emulsion: paraffin or kerosene oil $\frac{1}{2}$ to 1 pint, soap solution (soft soap 1 lb., water 1 gallon) 1 gallon. On the seventh day wash the animal. Flame the box with a brazier's lamp. On the eighth day sulphur dressing again. On the thirteenth kerosene emulsion. On the fourteenth day the animal is washed. Treatment is carried on for three weeks. Three courses of dressings are used. No bedding is permitted. The animal is kept under suspicion for three months afterwards. In-contact animals are dressed with the kerosene emulsion.

In the great European War, on account of the large numbers of animals affected with mange, individual attention could not be given to each case, and other methods of treatment have been tried, due regard being paid to economy of labour and the cheapness as well as the efficiency of the dressing. Where outbreaks have been large, special hospitals have been set aside for dealing with the cases, and special dipping tanks erected on the same lines as those used for dipping cattle and sheep. The common dip used is prepared from lime and sulphur. Unslaked lime is made into a paste with water and sulphur mixed in, in the proportion of lime 15 lbs., sulphur 20 lbs., water being added to 25 gallons. The mixture is boiled for two or three hours until the liquid becomes greenish yellow. The sediment is allowed to settle and the supernatant fluid is drawn off and water added to make 100 gallons of solution. The sediment is irritant and must not be used in the dip. The dipping tank holds about 3600 gallons of dip. This fluid is kept moderately warm when being used. After clipping and washing, the horses or other equines are passed through the dipping tank. This is repeated twice a week, and the dip rubbed in well after each immersion. A cure is rapidly effected with psoroptic mange, and good results have also been claimed with sarcoptic mange.

Last of all, Vigel and Chollet have introduced Lépinay's treatment of mange of the horse by sulphurous anhydride. A specially constructed air-tight box is used for the purpose. To it is attached an apparatus for the generation of sulphur dioxide. The horse is first clipped, washed, and dried. It is then backed into the box with the head protruding. Over the head is passed a cloth collar which is fixed, on the one hand, to the margins of the opening in the body through which the head protrudes, and, on the other, closely tied round the neck of the horse close to the head. This is done to prevent the escape of the fumes of sulphur dioxide and the gassing of the horse. The openings of the chamber are tightly closed and the gas turned on. As great a concentration as 6 per cent of sulphur dioxide is reached in the box. The animal is exposed to the action of the gas for two hours. Afterwards the ventilators of the box are opened and the gas escapes. The animal does not inhale gas at any time. The horse is then allowed to leave the box, and the head is treated with one of the ordinary dressings for mange, such as cresylated oil 1-10, and in psoroptic mange 3 per cent cresyl emulsion poured into the ears as recommended by Henry. This dressing is applied on several occasions. After exposure to the gas, the horse is put into a thoroughly disinfected stable, groomed daily, and washed on several occasions at intervals of a few days. A cure is rapidly effected (see p. 475).

Treatment of chorioptic or symbiotic mange is simpler than that of the two other forms in the horse. The hair should be clipped off from the limbs, and the limbs washed with soap and water and dried. Any of the dressings recommended in psoroptic or sarcoptic mange should then be applied on two or three occasions at intervals of a few days. Lime and sulphur dip, or 1 or 2 per cent creolin emulsion answer well. This is sufficient to ensure recovery.

Transmission of Mange of Equines to other Animals.—The Psoroptes or Chorioptes of the horse does not cause anything but a temporary irritation in other animals or man. The parasites rapidly disappear from the skin. The Sarcoptes of the horse may occasionally be transmitted to the dog or ox or man. It has been observed to affect soldiers or grooms who are often in close contact with affected horses, especially where no great attention is paid to washing the hands or arms after contact. The hands and arms are most frequently affected, but sometimes the disease extends to the chest and other portions of the body. Recovery may be spontaneous, but at times the disease is rather severe, and treatment is required before recovery takes place.

Follicular mange in the horse has occasionally been observed. It is due to *Demodex folliculorum equi*. There is no evidence that it is identical with that of the dog or man.

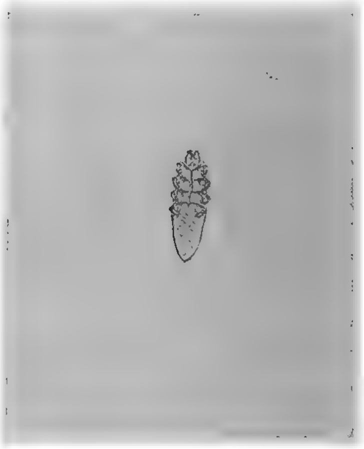


FIG. 119.—*Demodex folliculorum* var. *equi*.

It has been found by Wilson (1844) and Majouli (1897) in the eyelids, by Gros (1845) around the muzzle, and more recently by Walther, Schenzée, Berger, Meissner, Bidault, and Urbain.

In some cases the demodex appears to be responsible for skin lesions.

Symptoms.—The disease is met with in isolated cases. There is very little itching. The lesions appear on the eyelids, nose, or face. Occasionally extensive lesions have been observed not only over the head, but over the lateral aspect of the neck and trunk. The hairs over the affected area are raised up and cast off and scales or crusts cover the hairless skin. There are no nodules or pustules. Microscopic examination of scrapings of the affected areas reveals varying numbers of demodex. This parasite has also been found associated with sarcoptic mange.

Treatment.—A cure may be effected by any of the methods of treatment adopted against sarcoptic mange. In some instances treatment has to be applied over a period of several months. J. F. C.

Mange in the Ox

1. **Sarcoptic mange** is not so common as psoroptic mange. It is due to the *Sarcoptes scabiei* v. *bovis*. It is alleged to be transmitted also from the goat and pig.

Symptoms are rather similar to those of the horse. The animal frequently licks or rubs the parts affected. The disease may affect any portion of the body; the back, sides, croup, escutcheon, and udder are frequently first involved. The skin becomes nodular, scaly, denuded of hair in patches, thickened and wrinkled. The disease slowly extends and may affect a large proportion of the body. It causes loss of condition, and in the cow reduction of the milk supply.

2. **Psoroptic mange** is the most common form of scabies in cattle. It is due to *Psoroptes communis* v. *bovis*. It was studied first by Dorfeuille in 1813 and afterwards by Gohier, Delafond, and Gerlach. The ovigerous female measures 700 μ long by 450 μ broad, the male 550 μ long by 310 μ broad. The disease is rapidly transmitted from one animal to another, especially where they are closely housed.

Symptoms.—There is considerable itching, which induces frequent licking and rubbing. The lesions begin along the upper aspect of the body from the root of the tail to the poll, and extend thence over the sides of the neck and body. The legs are last involved. The lesions begin as little nodules which soon become hairless, and the skin becomes covered with thick scabs, thickened, scaly, and wrinkled, parchment-like and dry. The disease extends gradually, and causes loss of condition and even emaciation.

3. **Chorioptic mange or symbiotic mange** is not uncommon in cattle. It is due to the *Chorioptes bovis.*

The ovigerous female measures 385 μ long by 240 μ broad, the male 285 μ long by 210 μ broad.

The condition is only slightly contagious, and may affect only one or two animals in a herd. It is transmitted by contact, and by the litter. The disease is more marked in the winter.

Symptoms.—There is only slight pruritus. The lesions appear at the extremities of the limbs and the base of the tail. The hind limbs are more often involved and the affection appears on the fetlocks, pasterns, and even extends upwards as far as the hock. The lesions appear as thick crusts or wart-like growths. At the base of the tail bran-like scales are noted on the thin skin there. The parasites are often very numerous in the lesions. In very rare cases the disease may extend all over the body. The parasites are easily found in the crusts. As a rule, the condition is a very mild one, is more marked in the winter, and the condition of the animal is not markedly affected by this form of mange.

Treatment is on the same lines as the horse. Sometimes dips are used as for the sheep. Mercurial ointment must not be employed on account of the danger of poisoning from licking off the dressing. 2 to 5 per cent creolin or lysol emulsions are also used.

Differential Diagnosis.—The condition is recognized by the itching and general appearance and by the identification of the acarus. The acari are often numerous in the skin lesion. It can easily be distinguished from lice by the discovery of these parasites, and from eczema by the results of a microscopic examination of scrapings.

Follicular mange in cattle is not common. It is due to *Demodex folliculorum* v. *bovis*. This parasite is about 210 μ long by 55 μ broad. It has occasionally been observed in a large number of animals in a herd. It was noted first by Gros in 1845, later by Faxon in America, by Grim and Gehl, by Bugge in Germany, and by Griffiths in Nyasaland.

Symptoms.—As a rule there is not much itching.

The lesions appear first usually on the neck or shoulder and gradually spread to the ears, face, trunk, and upper part of the limbs. Gros saw the disease on the muzzle. The lesions have the form of round nodules varying in size to a hazel-nut. They contain a cheesy pus rich in demodex. Occasionally in the later stages after months they become reddened, denuded of hair, and rupture, giving exit to the cheesy pus. When the infection is a severe one in advanced cases the animals become emaciated, and some may even die of the disease, probably as the result of a toxæmia. The diagnosis is easy. The examination of the pus from the nodules reveals large numbers of the parasites. In many cases the disease is very mild, remains stationary, the hides only are affected, and the lesions are referred to as pimples.

Treatment.—Very often no treatment is adopted. It consists in segregation of infected animals, disinfection of the places with which they have been in contact, squeezing out of the contents of the nodules, and the application of one or other of the dressings recommended in mange in equines. Cooper's improved cattle dip 1-200 is also used as a local application, but it seems to be chiefly useful in preventing the further development of the disease and the spread to other animals. In extensive cases destruction is recommended. In any case animals affected should be sent to the butcher as soon as they are sufficiently fat for slaughter.

J. F. C.

Mange in Sheep

1. **Sheep Scab.**—The most important and common form of mange in sheep is *Psoroptic mange*, and is commonly referred to as sheep scab, although the latter term has been used to include the other forms of mange. One may say generally that when sheep scab is spoken of psoroptic mange in sheep is meant.

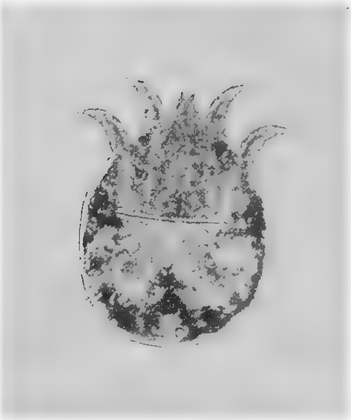


FIG. 120.—*Psoroptes ovis.* Pubescent female.

Cause.—It is due to *Psoroptes communis* v. *ovis.*

The female is 700 μ long by 455 μ broad, the male 550 μ long by 360 μ broad. The parasite lives on the skin of the sheep and does not thrive on the bodies of other species of animals. The psoroptes of the goat or rabbit will not live on the skin of the sheep beyond the second generation. Therefore, where a psoroptes is found on sheep, it may be taken as the *Psoroptes communis* v. *ovis.* The parasite lives and propagates on the skin and does not move out on the wool fibres more than one inch from the skin. It, however, reaches the surface when tufts of the wool fibres are cast off or rubbed off in the older lesions.

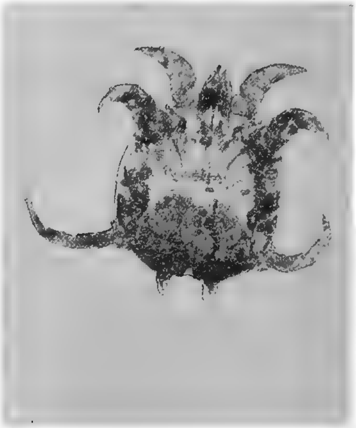


FIG. 121.—*Psoroptes ovis.* Male.

The acari may live off the host for twenty or thirty days, but only the ovigerous females live after about the tenth day. Eggs may retain their vitality off the host for about eight or ten days, but in ordinary conditions often fail to hatch out. It has been maintained by sheep-owners that fields and manure remain infective for a long time. That, however, is not borne out by experiment. Infective material removed from a scabby sheep and stored for twelve to fourteen days reproduced sheep scab (Stockman), but in no case has the infective material been shown to remain active for a longer period. Healthy sheep, put into

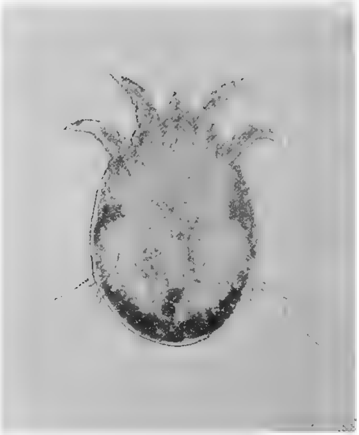


FIG. 122.—*Psoroptes communis* var. *ovis.*
Ovigerous female.

infected pens at varying periods after removal of affected sheep, became infected with sheep scab only when the intervals were not longer than eight or nine days.

Manure collected from a kraal in which infected sheep had been running for sixteen days was transferred to a clean kraal, and 4 clean sheep were admitted to the latter for five days. They became affected with sheep scab. When they were removed and clean sheep put into this kraal, no further infection resulted. This experiment only proved that the manure may attract the acari; it does not indicate that the acari will live long in the manure. These experiments go to show that the main source of infection is the infected sheep. An interesting point is raised by Henry. He found psoroptes in the ears of sheep which were emaciated but free from sheep scab in 15 out of 32 cases examined. He drew the conclusion that the normal habitat of psoroptes is the inner side of the ears, and that the parasites only occasionally leave the ears and set up body mange. I have been unable to corroborate Henry's findings, but I found psoroptes in the inside of the ears of three sheep badly affected with sheep scab out of a flock of ninety-five sheep in which the disease had broken out. The results of investigation, however, point to the importance of the treatment of the ears in sheep scab.

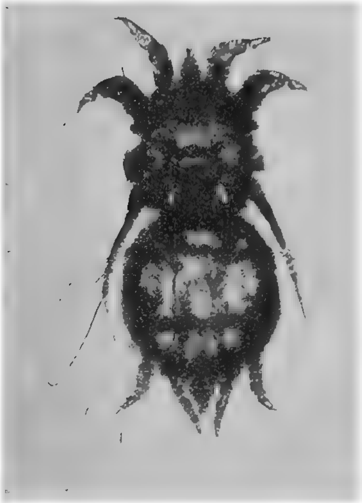


Fig. 123.—*Psoroptes ovis.* Male and female *in copula.*

Transmission.—Sheep scab is the most serious and most contagious form of mange in sheep. It sometimes causes serious losses and brings about considerable depreciation in the value of the fleece. It is easily transferred by contact, and through common rubbing-posts, hedges, or bushes, through public conveyances, railway wagons, markets, or fairs. Other parasites in sheep may facilitate the transmission, because of the use of common rubbing-posts. The transmission is easiest in proportion to the degree of close association of the sheep.

It is a remarkable fact that in the British Isles, at all events, outbreaks of sheep scab, like some other forms of mange, are much more numerous in the winter months. This may be due to several factors. There is a considerable amount of traffic in sheep in the autumn, and thus sheep scab may be more widely disseminated at this period. Summer dipping also is a factor in the decrease of outbreaks in summer. The sheep scab parasite probably thrives better under certain conditions on the skin of the host. Shilston has shown that dry weather causes a marked decrease in the reproductive activity of the psoroptes, and on skin covered with a thick layer of yolk they may eventually die off. No doubt also the long fleece of the sheep in winter favours the development of the parasite. The period which elapses after infection until noticeable symptoms appear conforms with what has been already written in connection with mange generally. It is generally accepted that in the average cases symptoms may be observed within twenty-five days after infection. But in some cases the period is increased to two, three, and four months, especially if the parasites which have passed on to the sheep are few in number or if the animal has been previously dipped. The reappearance of scab in sheep several months after apparent recovery as the result of dipping has frequently been noted. Thus in some experiments of Stockman, sheep infected with three female psoroptes showed symptoms only after seventy-seven days; and sheep after being dipped and then infected with twenty copulating pairs and twenty female psoroptes presented distinct symptoms only after two hundred days. Again it has been observed that lambs running with affected ewes usually get acari on their skin, but although the parasites seem to flourish and breed the symptoms of sheep scab do not appear on the lambs for months.

Distribution.—Sheep scab is found in all countries wherever sheep are reared. The disease, however, has been completely eradicated from Australia and New Zealand. It has been recognized from ancient times, and although it has caused serious losses and very extensive outbreaks it has not received, save in the countries mentioned, the attention it deserves so as to bring about its complete eradication. This is probably due to slip-shod methods or perfunctory actions which are adopted.

Symptoms.—One of the marked symptoms of the disease is pruritus. This is particularly marked when the sheep is heated from exercise or kept in a warm place, or when the sheep are crowded together. It is not very evident when the animals are exposed to cold wet weather or have been douched with cold water. The sheep frequently rubs itself against posts or trees or fences, sometimes scratches the part with the limbs, or nibbles or bites it. Sometimes it may even pull out locks of wool. If the affected area is rubbed the sheep stretches out its head, turns round and nibbles with its lips.

The parts of the skin affected generally are those covered with fleece. Those first involved are often the back and withers, the tail and scrotum in lambs, occasionally the under aspect of the breast, but any part of the body may be attacked. It is only in extreme and exceptional cases that the disease extends to the limbs.

The early lesions often escape detection because of the necessity of a very careful examination of the body and the difficulties of inspection resulting from the presence of a long fleece. The early stage consists of little red papules, where the parasite has bitten, no larger than a pin-head. Soon a little serous fluid exudes and coagulates at the base of the wool fibres. This process is accompanied by proliferation of the epithelium, and the surface of the nodule becomes covered with silver grey scales under and between which the serous fluid forms a wax-like material. The lesions may be multiple and fuse, and may extend in a centrifugal or peripheral, rarely linear, fashion. It is usually roughly circular, and soon attains the size of a sixpenny or a shilling piece. When the parasite has caused the lesion it passes to a fresh normal area of skin. In the older lesions the central portions become dry, and the wool falls out in tufts or is rubbed or torn out. The tufts contain at their base the yellow wax-like material which agglutinates the fibres and is rich in psoroptes. The bare patches of skin become dry, thickened, and parchment-like, and covered with large greyish scales or crusts. These contain very few parasites. As the result of this process the fleece becomes broken and ragged, and the wool fibres dry and brittle and depreciate in quality. Redness and abrasions of the skin sometimes result from rubbing, and occasionally small pustules or abscesses form.

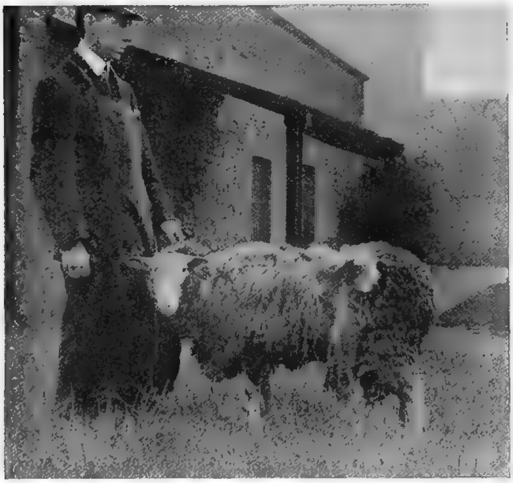


FIG. 124.—Sheep affected with sheep scab.

On account of the restlessness caused by the action of the irritating material injected by the parasite the sheep loses condition and may become emaciated. In neglected cases emaciation and debility become extreme after a varying time, and death may result from sheep scab. If the animal survives, recovery from scab takes place usually after the disease has spread all over the body, head, and limbs. Should the disease recur it begins again at the tail or head. It has also been noted that shearing scabby sheep retards the progress of the disease.

Diagnosis.—When a flock of sheep is being inspected for sheep scab a ragged fleece and evidence of pruritus are suspicious symptoms. In the early stages the fleece may appear normal. To bring out symptoms of itching, the sheep may be huddled together for a time in a warm shed or given some exercise and then put into a pen, when affected sheep will begin to rub against posts or fences. The suspicious sheep are then caught and examined for lesions. In the early stages a close inspection must be made of various parts of the body, including tail, scrotum, and under aspect of the chest, before a lesion is found and a reaction obtained by scratching the part. Diagnosis is made certain by the discovery of the parasite. Scrapings are best taken from the early lesions, and the best material for examination is the yellow wax-like material which is found at the base of the wool fibres in the lesions. This material may be so rich in parasites that they may be recognized with the aid of a small hand lens, or it may be subjected to any of the methods already described for the discovery of mange parasite in scrapings.

Differential Diagnosis.—Other animal parasites causing pruritus of the skin include sheep ked (*Melophagus ovinus*), sheep lice (*Trichodectes sphærocephalus*), maggots, harvest bugs, and ticks.

The sheep ked causes only slight annoyance, and is easily detected in the fleece. Sheep lice cause as severe trouble as sheep scab, and are differentiated by the recognition of their presence. Ticks may also cause itching, but are easily discovered. Maggots most commonly affect the croup or the base of the tail in young sheep, and when nests of them are touched the animal smacks the lips vigorously. The scurfy condition of the skin which is left by maggots often causes some trouble in differentiating from sheep scab. Harvest bugs (the larvæ of the *Trombidium holosericum*) appear in the autumn on certain pastures and attack the under aspect of the body. They may be so numerous as to give a rusty appearance to the wool fibres. They are easily detected, and the orange-red hexapod larvæ are easily distinguished from the sheep scab parasite.

Excoriation of the skin may result from dipping with too strong a dip, *e.g.* an arsenical dip. Parts of the skin become hard and necrotic, and patches become detached with a tuft of wool fibres attached. The fleece may thus appear somewhat ragged, but there is no itching.

A condition called **Rain-scab** has been observed in sheep in the wet season. The skin becomes very scaly and the wool dirty-looking, especially over the back. The sheep have rather a shaggy appearance, but there is no pruritus.

In some of the fine-wooled sheep, where the wool is thick and close, there is often a thick layer of yellow soft yolk at the base of the wool fibres. Sometimes such sheep react to scratching, but the skin is quite clean, and there are no parasites.

Last of all, an occasional sheep may show signs of pruritus without any signs of skin lesions. Such sheep have been spoken of as nibblers; they are not affected with sheep scab.

Treatment.—Sheep scab in the British Isles is a scheduled disease dealt with under the Diseases of Animals Acts. An outbreak of the disease must be notified by the owner or shepherd and by the veterinary practitioner who is called in to see the animals.

Restrictions are placed upon the whole flock, although some of the animals may appear healthy. These latter are treated in the same way as the affected sheep, because no one can be sure that they are free from sheep scab. The sheep must be confined to certain folds or enclosures, and must not be allowed to pass along or across highways.

They are then dressed with some parasiticide dressing. Previous to dressing they should first be clipped and the affected areas scraped to permit of intimate contact of the dressing. This is done not only in summer but also in winter. In-lamb ewes must also be dressed; any damage which may be done to the ewes is not due to the dip, but to the handling of the animals.

The dressings are applied usually in the form of dips. Various dips are employed, and are mostly proprietary preparations. They consist of a variety of agents, including lime and sulphur, carbolic acid, creolin, arsenic, and tobacco decoctions.

Examples of some of the preparations used are:

1. *Lime and sulphur dip,* prepared in the same way as that described for mange in equines.
2. *Arsenical dip:*

Arsenic.
Pot. Carb.
Sulph. Sub. āā lbs. ii.
Sapo Moll. lbs. iv.

to be dissolved in 10 gallons of boiling water and made up to 100 gallons. An arsenite of soda dip is also used, consisting of

Arsenite of Soda	2¼ lbs.
Sulphur	5 lbs.
Water	100 gallons.

3. Creolin or cresyl in 2 to 2½ per cent emulsion in water.
4. *Carbolic Acid Dip:*

Acid. Carbolic.	3 quarts.
Sapo Moll.	5 lbs.
Water	to 100 gallons.

5. *Tobacco Decoctions.* — Standardized in America so that they contain not more than ·07 per cent nicotine. This is necessary to avoid losses from nicotine poisoning.

A home-made preparation consists of 30 lbs. shag tobacco steeped overnight in 30 gallons of water, brought to boiling-point in the morning, allowed to simmer for a few minutes, strained through a sack, and left overnight. It is then made up to 100 gallons. The preparations must not be made too strong, and must be carefully mixed.

The dip used should be lukewarm when run into the dipping-tank. Many forms of dipping-tank are used, and in all cases should be sufficiently deep to allow of complete immersion of the sheep. In the dipping process the sheep should be kept in the tank for two minutes and completely immersed at least twice. Special attention must be paid to the head, neck, and tail to ensure that they are properly dressed. Afterwards the sheep are passed on to a strainer, and the excess of dip squeezed out, the excess draining off into the tank. Then the sheep is put into an enclosure free from fodder until it is dry. This is done to prevent poisoning from contamination of pasture or fodder with the dip which drips off the fleece or bodies of the sheep. Afterwards the animals are allowed into a grazing paddock. In a large flock dipped sheep may be allowed in contact with undipped provided (*a*) all sheep are dipped within a few days of each other, and (*b*) a second dipping of all sheep is carried out within eight days of the first. One dipping is not sufficient to bring about a complete cure. Very few dips destroy the eggs (Shilston points out that the lime of sulphur may prove effective in this way), and a few adult parasites may survive. A second dipping must therefore be carried out within eight days of the first in order to prevent a second generation of the parasites being produced. A third dipping within a month is also necessary for some cases, especially when they are advanced. The inner side of the ears should also be wiped out with the dip, or, as Henry recommends, a small quantity of 2 to 3 per cent warm watery emulsion of cresyl poured into each ear.

Where dipping-tanks are not available the sheep may be dipped in an ordinary tub filled with the dip, or hand-dressed.

Special precautions are taken to avoid losses from poisoning by arsenical dips. The sheep should not be dipped on a very hot day. They should be rested for some hours after being driven to the dipping tank before they are dipped, and they should not be driven long distances for twenty-four hours after they have been dipped. Affected sheep with large wounds should be carefully hand-dressed to avoid any danger of poisoning.

During the course of the treatment the sheep should be well fed.

Re-infection from the land on which the scabby sheep have been grazing is a minor point which should receive attention. Loose wool on the ground, on bushes or fences—manure in the folds should be gathered together and burnt. Rubbing-posts, fences, trees should be smeared with tar, lime-wash, or dip to destroy the acari. Experimental evidence has, however, shown that these places remain sources of infection for only a short time.

The sheep must be kept under restrictions until they are free from infection, the itching has ceased, the scabs have disappeared, the skin is normal, and the fleece growing well. A very careful inspection must be made before the sheep are regarded as cured. It would be well if restrictions are maintained for at least two or three months, because evidence of recurrence may only be shown in that time. The sheep may be only partially cured, and if restrictions are taken off the sheep farmer may sell his sheep in the market, and sheep scab may be distributed among other flocks.

Prevention.—Sufficient powers have been obtained in the British Isles under the sheep-dipping orders to bring about a considerable decrease in the number of outbreaks, provided the orders are uniformly and rigorously carried out with the co-operation of flock-owners.

Sheep must be dipped on two occasions yearly, in the early summer after shearing and in the autumn. On the second occasion it is required that the sheep must be dipped twice at an interval of eight days. Movements of sheep are not permitted till this is done. Sheep scab is kept going through defects in the carrying out of the regulations. In the rounding up of sheep, some of them may be missed on large hill farms, and hence flocks in mountain districts are a frequent source of sheep scab. Dipping to be effective must be thoroughly carried out, and fines of sufficient severity imposed to ensure this being done. Owners of sheep should safeguard themselves against the admission of the disease to their flocks by first dipping all new sheep, and even their own sheep returned from the market, before mixing with the others on the farm. Trucks, sale-yards, enclosures at public fairs must be frequently disinfected.

2. **Sarcoptic mange** in sheep is very rare. In the British Isles it has occasionally been noted in Scotch black-faced mountain sheep.

It is due to the *Sarcoptes scabiei* var. *ovis.* In this variety the female is about 400 μ long by 300 μ broad, the male about 210 μ long by 160 μ broad. This acarus according to Railliet may develop also on the goat, and it is only rarely transmitted to man.

This form of mange in sheep is not nearly so serious or so contagious as psoroptic mange.

Symptoms.—The disease is accompanied by a severe pruritus which causes the animal to rub and scratch the affected parts. The affection is chiefly confined to the head (hence the term head mange applied to the disease). It does not affect the parts of the body covered with wool. It begins often about the lips and wings of the nostrils, sometimes the ears and eyelids, and thence extends to other parts of the head. Little papules first form on the skin, then the hair is cast off from over these papules. The skin becomes scaly and covered with light coloured or grey crusts of epidermis. The lesions, at first separate and irregular, become confluent. If the crusts are removed, a raw red surface is exposed. The disease is slowly progressive and causes loss of condition. It may be complicated with conjunctivitis. Ulcers and scabs are produced from the rubbing.

Treatment.—The animals are treated on the same lines as sarcoptic mange in the horse, or with sheep dip at intervals of three or four days, until a cure is effected. The disease responds readily to treatment. Measures of isolation and disinfection are applied as in psoroptic mange.

3. **Symbiotic or Chorioptic Mange in the Sheep.**—This is a comparatively mild affection and only sometimes requires attention.

Cause.—It is caused by *Chorioptes ovis.*

The female is about 385 μ long by 240 μ broad, while the male is 310 μ long by 250 μ broad.

The disease is not very common, and only slightly contagious. Only about 1 per cent of the flock may be affected.

Symptoms.—The disease affects the limbs almost solely, most commonly the hind limbs. There is some pruritus, the sheep sometimes rubbing or gnawing or stamping the limbs. The lesions begin in the hollow of the pastern and extend upwards. In neglected cases they may reach the scrotum or mammary gland. Sometimes it affects only the interdigital space and causes lameness. The skin on which the parasite develops is at first red and afterwards denuded of hair. Then greyish crusts form which vary in thickness and may present a wart-like appearance.

The parasite can easily be found in scrapings of the lesions.

Symbiotic mange is most marked during winter and tends to disappear in the summer months.

Treatment.—The only treatment necessary is to pass the animal through a leg bath of sheep dip on one or two occasions, the second bath being given after a week's interval.

Mange in Goats

1. **Sarcoptic Mange** is the most common and serious form of scabies in goats.

It is due to the *Sarcoptes scabiei* var. *capræ*.

The female measures 345 μ long by 342 μ broad and the male 243 μ long by 188 μ broad. This variety may be transferred to the sheep, ox, horse, pig, and even man. This disease has been observed in France, in Africa and Asia, but no case of it has been recorded in the British Isles. It is highly contagious and rapidly transmitted directly or indirectly through a herd.

Symptoms.—The disease is accompanied by great itching. Lesions begin usually on the head and ears, thence pass to the neck, to the chest and abdomen, even the udder, and last of all extend to the limbs. They appear first as small nodules from which a serous fluid exudes and coagulates on the surface. The epidermis over the nodules forms a dry, scaly crust which is cast off with the overlying hair. The skin in the hairless patches becomes thickened, and scaly, rugged, cracked, and grey in colour. The patches afterwards coalesce. Numerous sarcoptes may be found in the crusts.

The disease if neglected becomes generalized, causes rapid emaciation, and even death.

Treatment is similar to that for sarcoptic mange in the horse. Measures of isolation must be adopted. Sheep-dip may be used as in sheep scab, but goats do not stand dipping well.

It may be more economic to destroy animals extensively affected with the disease than to treat.

2. **Psoroptic Mange in Goats.—Psoroptic Otacariasis in Goats.**—This disease is due to the *Psoroptes communis* var. *capræ*, a parasite similar to psoroptes of sheep. The disease set up by the parasite is highly contagious, and appears to be common only in certain districts or countries.

Symptoms.—Otacariasis is confined to the inner side of the external ear. The skin there becomes covered with brown crusts, which may also fill up the opening. The parasites are present in abundance on and in these crusts. There is some itching and the animal sometimes rubs the ear or scratches it with its hind limbs. In some cases the affection induces some decrease in appetite; it seldom causes death.

Treatment consists in the isolation of affected animals, removal of the crusts from the ear, and the application by pouring or smearing of some parasiticide dressing into the ear, such as 2 to 2½ per cent creolin or cresyl, or a watery warm emulsion of petroleum 1 part, cocoa nut oil 2 parts. One dressing may be sufficient to effect a cure.

3. **Symbiotic or chorioptic mange in goats** is not a common affection. It has been seen in Angora goats. It is due to *Chorioptes capræ*. The female measures 340 μ long by 220 μ broad, the male 295 μ long by 295 μ broad.

Symptoms.—The disease affects the side of the neck, behind the ears, on shoulders, back, and loins, and is accompanied by slight itching. There is an incomplete loss of coat, the skin becomes scaly, thickened, and wrinkled, and covered with yellow coarse crusts.

The disease progresses very slowly and is benign. The parasites are easily discovered under the crusts formed on the skin.

Treatment is as for sarcoptic mange.

4. **Follicular mange in the goat** is rare. It is due to the *Demodex folliculorum* var. *capræ*. The disease appears in the form of small nodules about the size of a pea or slightly larger, which develop on the back and side. From the nodules may be squeezed out a cheesy pus which is composed largely of the parasites.

Mange in Camels

Sarcoptic mange is the only form of scabies affecting the camel, dromedary, and other animals belonging to the Camelidæ.

It is due to the *Sarcoptes scabiei* var. *cameli*.

The disease is very contagious, and easily transmitted by direct contact and by intermediate carriers. Its spread is favoured by close associations of large numbers of animals, as when they are used for transport. It is more common and progresses most rapidly in the young and very old animals, and is often associated with debility and dirt. It may be transmitted to man.

Symptoms.—A most marked symptom is pruritus, the animal frequently rubbing and biting the parts. The disease begins where the skin is thin, the under aspect of the abdomen and the inner side of the thighs, and it rapidly extends over the body to the withers, tail, and the limbs. First there appear small nodules from over which the hair falls out. The hairless spots extend and fuse and the skin becomes thickened, cracked, wrinkled, and scaly or covered with crusts. As the results of rubbing, ulceration of the skin occurs in places.

The disease progresses rapidly, causes wasting, debility, and even death.

Treatment is on the same lines as for sarcoptic mange in the horse. A parasiticide dressing commonly used consists of sulphur, salt, āā ½ lb., train oil 2 lbs. Abundant food forms an essential part of the treatment, and light exercise should be given. The natives sometimes treat the animals with daily sea baths. After immersion in the sea, the skin of the camel is energetically scrubbed with a smooth stone. A popular dressing also used consists of tar 2 parts, water 1 part.

Mange in the Pig

1. **Sarcoptic mange** in the pig is due to *Sarcoptes scabiei* var. *suis.* This is the largest of the varieties of Sarcoptes. The ovigerous female is about 450 μ long by 360 μ broad, the male is about 300 μ long by 250 μ broad. This sarcoptes may be transmitted to man.

This disease is not common. It is slowly contagious and may pass through a litter of pigs. It affects young pigs most severely. Its progress is most rapid when it is associated with insufficient nourishment and debilitating diseases. It is often difficult to discover the source of infection among a herd of swine.

Symptoms.—The first symptom noted is a most intense pruritus. The animals are constantly rubbing themselves against posts, walls, their troughs, or along the ground or floor. They frequently scratch the abdomen and inside of the thighs. The parts of the body rubbed or scratched become red, denuded of bristles, and are marked by scratches or abrasion.

The lesions begin frequently on the ears, along the abdomen, and inside of the thighs. They have the form of little firm nodules, which may be very numerous, but separate and present a little central point, and the skin around them is red. They soon become covered with small grey scabs or crusts and the bristles fall out. Later, the lesions fuse, become very large, the skin becomes thickened, dense, and leather-like, covered with thick fissured scabs, and form thick folds or cracks. The larger portions of the head, body, and limbs become involved, and the skin in addition to being thickened and wrinkled appears to be covered with a light grey powder. When the disease becomes extensive, the animal becomes very much emaciated and weak. The removal of the crusts exposes a raw red surface.

When the pigs are fed well and kept clean, the disease may spontaneously disappear in the summer after one, two, or three months. The progress of the disease is generally very slow. Sometimes it remains localized and does not interfere materially with the fattening process.

The diagnosis is easily made. The parasites can easily be detected in the crusts formed in the lesions. The condition is readily distinguished from eczema of sucklings and from the affection caused by lice (*Hæmatopinus suis*).

Treatment is similar to that for sarcoptic mange in the horse. The thick crusts must first be removed by scraping and washing. Sulphur dressings are very efficacious. With lime and sulphur dip the deposit, as well as the liquid, may be used without ill effect. The disinfection of the styes must be carefully carried out.

Follicular mange in the pig is not common; it has been described on the Continent, Africa, and in America. It is due to the development in the hair follicles and sebaceous glands of *Demodex folliculorum* var. *suis* (*Demodex phylloides*). It is thicker than the demodex of the dog. The female is 250 μ long by 63 μ broad and the male 220 μ long by 55 μ broad. The disease is only slightly contagious and does not affect the general health of the animal.

Symptoms.—There is little pruritus. The lesions have the form of nodules or pustules which develop in the skin where it is fine, chiefly on the snout, neck, under aspect of the chest and abdomen, and inner side of thighs. The nodules are about the size of peas, sometimes pigmented and deep-seated, and often surrounded with a red zone. The pustules may contain very large numbers of parasites, from 50 to 1000. Sometimes they rupture and leave large ulcers.

No *treatment* is adopted, as the disease does not interfere with fattening but affects the condition of the hide. It does not affect the suitability of the flesh for the food of man.

Mange in the Dog

Sarcoptic mange is the most common of all the skin diseases in the dog.

The cause was first demonstrated by Gohier in 1814 and has since been described by Gurlt and Hertwig, Gerlach, Delafond, Megnin, and others. It is the *Sarcoptes scabiei* var. *canis.* The adult female is about 350 μ long by 260 μ broad while the male is about 210 μ long by 150 μ broad. Occasionally this parasite is transmitted to man, and according to Zurn, to the pig and horse. I have also observed a few cases of mange in the dog due to the *Notœdres cati*, probably due to infection from the cat.

Occurrence.—Sarcoptic mange affects dogs of all breeds and various ages. I have seen the disease in puppies three or four weeks old.

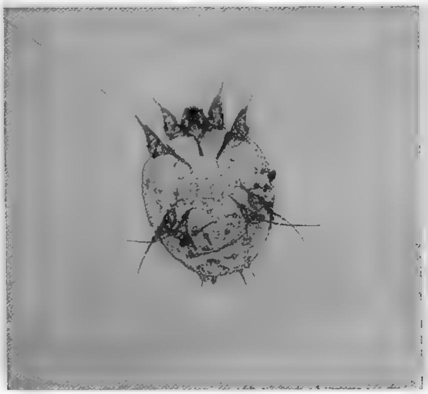


Fig. 125.—*Sarcoptes scabei* var. *canis*. Ovigerous female.

Transmission.—The disease is highly contagious, is transmitted chiefly by direct contact with affected animals, but also to a lesser extent from infected litter, by introduction to infected kennels, by attendants, by clothing and brushes. The disease is commonly kept going in breeding and dealer's kennels, where the condition is frequently referred to as eczema, and by dog shows. Animals frequently groomed and washed are not so liable to become infected.

Symptoms.—There is marked pruritus. The dog frequently scratches itself with the hind or fore limbs, rubs its body against neighbouring objects and bites itself. When the affected area is scraped, the dog makes vigorous scratching movements with the hind limbs. The intensity of the pruritus varies with individuals, is greater in older animals, and is most marked when the dog is warm.

The first part usually invaded is the head. The lesions there appear around the eyes, on the face, and the outer side of the ears. Then they develop along the under aspect of the neck, the under aspect of the abdomen and chest, on the hind and fore limbs, especially on the outside of the elbows and above the hocks. In severe and old-standing cases the disease involves the back and ultimately the whole of the body.

In the early stages the epidermis over small areas becomes roughened, lumpy, thickened, and scaly, and little tufts of hair are cast off. The patches are irregular and increase in size at the edges and by fusion. Little nodules about the size of a pin's head may sometimes be seen and felt. When the skin is unpigmented, it becomes slightly reddened, and a little dark spot may be seen on the centre of each nodule. This is most commonly noted on the abdomen and side of the chest. Occasionally small pustules about the size of millet seeds develop, especially on the abdomen. Later, the skin becomes scaly and large thin epithelial flakes are cast off or remain adherent to the overlying hairs which are not as yet detached. Where the skin around the eyes is involved and denuded of hair, the animal appears lemur-like. The eyelids may become thickened and slightly inturned, and symptoms of conjunctivitis result. In advanced cases the large proportion of the coat is cast off. On account of the scratching and biting, some areas of skin, especially over elbows, under abdomen and chest, and at side of head and base of ears, become reddened, moist, and ulcerated, and ultimately covered with dried blood and black scabs. As the disease advances, the dog loses condition, becomes emaciated and weak. The skin is greatly thickened and wrinkled. Even death may result from exhaustion. The progress of the disease is retarded by good grooming, frequent washing, and generous diet, but is hastened by debility, debilitating disease, such as distemper, and by bad hygienic conditions.

Diagnosis is made from the appearance and position of the lesions of the skin, the pruritus and the discovery of the parasite. The latter is carried out by the method described for mange generally. Scrapings are best taken as a rule from the outer side, and towards the tip of the ear and the front of the forehead.

Differential Diagnosis.—The disease is thus differentiated from lice, the eggs and adults of which can easily be discovered (it must be remembered, however, that lice sometimes complicate sarcoptic mange), from attacks of the harvest bug, from the squamous form of follicular mange (in the latter cases there is a peculiar odour from the skin, pruritus is not marked, and the parasite is differentiated in scrapings), otacariasis (the disease here affects only the inner side of the ear. It may com-

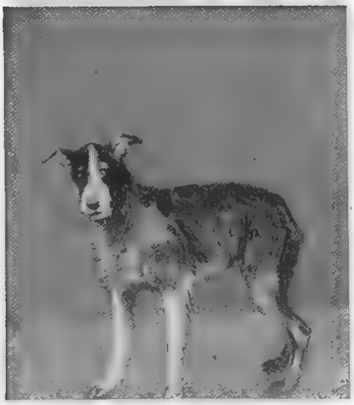


Fig. 126.—Dog affected with sarcoptic mange.

plicate sarcoptic mange), dry eczema (many of the cases of which as diagnosed are really cases of sarcoptic mange), pruritus of the back and loins commonly seen in adult and old dogs.

Treatment is identical with that recommended for the horse. It is usually necessary to clip the dog all over. Sulphur, oil of tar, potassium bicarbonate and lard dressing as noted for the horse are extremely useful for the dog, and applied in exactly the same way. Attention is paid to isolation, changing and burning bedding weekly, and periodic application of disinfectants to the floor and walls of the kennels. In cold weather the animal must be kept warm, and in all cases must be fed well. Liquor arsenicalis given internally may be useful where the disease is advanced. In average cases a cure may be effected in a month if the treatment is thoroughly carried out.

Another useful dressing is

Oil of tar.
Soft soap āā 1 part.
Meth. spirit 8 parts.

This is applied every three or four days and three or four dressings are often sufficient. The skin, as the result of the application, becomes at first very red as well as scaly.

Petrol applications are good. Lime and sulphur dressings as recommended for the horse are used in the dog. Balsam of Peru 1 part, meth. spirit 5 parts, is also employed, is rather expensive, but is not so objectionable as the other dressings when applied at home to house dogs. Mercurial and carbolic dressings must be avoided on account of the danger of poisoning from licking off the dressing.

After recovery the kennels are disinfected with boiling water or 2 per cent creolin or other reliable disinfectant, and the utensils, brushes, clothing, and collar boiled or immersed in some reliable disinfectant fluid.

Sarcoptic mange is also noted in foxes and other wild carnivora, and is similar in its appearance to that disease in the dog. Destruction of the affected animal is often the most advisable method of eradication.

2. **Otodectic or Symbiotic Mange in the Dog. (Otacariasis, Parasitic Canker of the Ear.)**—This disease is due to *Otodectis cynotis* var. *canis* (*Symbiotes auricularum*) first noted by Hering in 1834, afterwards studied among others by Megnin and Nocard.

The ovigerous female is 500 μ long by 310 μ broad, the male 365 μ long by 265 μ broad.

The disease is noted in various breeds of dogs, including sporting dogs, setters, pointers, hounds, terriers. I have observed it most commonly in Pomeranians.

It is highly contagious and spreads rapidly from dog to dog by close association. Sometimes it is associated with sarcoptic mange.

Symptoms.—The disease is confined to the ears. The affected animal frequently scratches and shakes the head. On account of the rubbing and scratching the hair is partly removed from the outer side of the ear and the side of the forehead, and some abrasions may result. The dog presses on the hand that rubs the ear. When the interior of the ear is examined, it will be seen that the skin towards the depth is covered with a brownish semi-solid discharge across which little grey specks are moving. These are the parasites. The skin there is not reddened, painful, or ulcerated.

In some cases fits and rolling movements have been observed in affected dogs. In hounds, when exercising, fits were noted by Nocard as the first symptoms of the affection. Complications produced by the disease from the shaking of the head and scratching are ulceration of the edge of the external ear and othæmatoma. Deafness has also been noted. The itching may cause serious loss of condition.

Diagnosis.—The parasite can, as a rule, be easily found in the wax-like material removed from the ear and identified under a low power of the microscope. The otodectes pass downwards as far as the tympanic membrane, hence are difficult to reach with a parasiticide dressing. Some of the parasites may leave the inside of the ear and appear on the surface of the skin in the neighbourhood, thus permitting of their easy transference to another animal.

Treatment.—The affected animal must be isolated. The discharge in the ear is first softened with some solution such as methylated spirit (1 to 4 of water), peroxide of hydrogen, or soap solution, and then carefully removed with plugs of cotton-wool applied at the end of a penholder or a pair of forceps. This should be done every week. Then a parasiticide dressing is inserted into the depth of the ear, such as sulphur ointment, salicylic ointment (1-8), or mercury nitrate ointment 1 part, olive oil 8 parts ; this should be applied every second or third day until the disease disappears. The outer side of the ear should also be dressed. Two per cent warm watery emulsion of cresyl may be employed. Nocard used a dressing of

Naphthol B	1 part.
Ether meth.	3 parts.
Olive oil	10 parts.

The material is poured into the ear, and the latter is kept plugged with cotton wool for about ten minutes to prevent evaporation of the ether. Then the animal is set free. This treatment is carried out daily. The ether is a solvent for the cerumen and allows of the penetration of the naphthol B, which acts on the otodectes.

Follicular Mange.—Demodectic mange is one of the most serious and obstinate skin diseases of the dog. It is due to the *Demodex folliculorum* var. *canis*.

This parasite was first found by Topping and described by Tulk in 1843. The female measures 390 μ long by 45 μ broad, while the male is about 230 μ long.

It invades the hair follicles and sebaceous glands of the skin, where it may be found in all its stages. The anterior end of the parasite is usually directed downwards. From 20 to 200 may be found in one hair follicle. In invading these structures the demodex carries with it the *Staphylococcus pyogenes albus*, which is inoculated into the tissues of the skin and sets up a dermatitis. In the pustular forms these cocci form small abscesses or pustules. The disease is fairly common, is met with in all breeds of dogs and is particularly frequent in bulldogs. It is quite common in Irish terriers. It may appear in a litter of puppies, but only one or two of them become affected. The disease appears in dogs during their youth, but is also seen in adults.

Follicular mange is only slightly contagious. It is only very occasionally that indubitable evidence can be brought forward of the transmission of the disease from one dog to another. Attempts at experimental transmission have not been successful. Sometimes the disease appears in certain strains of dogs. Probably there is some special idiosyncrasy or susceptibility inherited or acquired necessary for the development of follicular mange.

Symptoms.—There is very little pruritus in this disease, but the dog frequently shakes itself. The skin usually gives off a characteristic sulphur-like odour. In some cases it is dark pigmented, while in others it is reddened.

The lesions often appear in the same positions as sarcoptic mange, but may develop on any portion of the body.

Two varieties are distinguished, pustular and squamous. In the squamous form the skin is usually pigmented; it becomes thickened and scaly in patches, and the hair falls out. When these patches are washed, little nodules over the hair follicles may stand out prominently. In a few cases the bare spots take on a circular form (circinate variety) which present some resemblance to the lesions of ringworm. The patches sometimes become very large, and a large proportion of the body is involved.

The dog may remain in fair condition, but the disease is chronic; it is less difficult to deal with than the purulent form. The diagnosis in this case is made by taking scrapings from the skin as in sarcoptic mange, soaking them in caustic potash or soda solution, and examining them in a thin film on a slide under a low power of the microscope.

In the pustular form the skin becomes reddened, even copper red, hot, and scaly in patches, and the hair falls out in tufts. A circular arrangement of the lesions is sometimes noted also in this form. Small nodules and pustules form over the hair follicles. These are at first about the size of pin-heads or a little larger, and the tops of the pustules are white or greyish in colour. From them a little thick, white or cheese-like pus may be squeezed out, which when examined in a thin layer on a slide under the low power of microscope will be seen to contain quite a number of specimens of the *Demodex folliculorum*. Sometimes the pustules increase in size and are transformed into large abscesses, which are bluish or purple on the surface, are easily ruptured, sometimes rupture spontaneously, and give exit to a blood-stained pus. This pus contains few specimens of demodex, but in films from it staphylococci may be easily identified after appropriate staining. On account of the burrowing of this pus, the skin involved is transformed into a sponge-work.

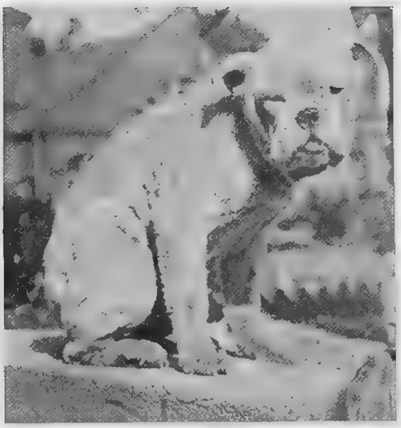


Fig. 127.—Bull-dog affected with follicular mange.

The disease in these latter cases often extends rapidly over the body and limbs. The skin becomes red, moist, and thickened, and forms thick wrinkles or folds.

As the result of a general coccus infection a toxæmia is rapidly produced, the temperature rises to 103°-105° F., the dog becomes very dull and weak, feeds badly, and rapidly looses condition. It may become emaciated within a week, and death in some cases rapidly follows.

The diagnosis is made from the absence of pruritus, the smell and characters of the skin lesions, and by the discovery of the demodex on microscopic examination of the scrapings of the skin in the squamous form or the pus in the pustular form. The pustular form is thus distinguished from acne and the squamous form from sarcoptic mange, dry eczema, alopecia, and erythema.

The *prognosis* of follicular mange should always be guarded. The cases may be under treatment for three, six, twelve months or more, sometimes they appear to recover, but the disease often recurs when treatment is stopped.

Treatment is not hopeful. There is no specific treatment. Large numbers of methods and applications have been adopted from time to time; a few cases may recover, and the method then enjoys a brief popularity. It is difficult to reach the parasites on account of their deep position. If treatment is adopted, the animal must be fed well. Autogenous vaccines as recommended by Professor Mettam have yielded some successful results. Nuclein has been adopted in its stead and appears useful in some cases. It is given hypodermically in the proportion of 1 minim per lb. body weight once a week.

The body or area affected is clipped, better shaved and washed, the pustules squeezed out in the pustular form, and the skin dried well. Iodine preparations in the form of ointments or liniments such as Tr. Iodi are often useful applied twice a week. The same preparations which are used for sarcoptic mange have been tried in follicular mange with varying results. Hunting recommends a dressing of—

Creosote	1 part.
Liquor Potassæ	2 parts.
Ol. Olivæ	14 parts.

applied every third or fourth day.

Almond used successfully a 2½ to 5 per cent zinc chloride solution in water. After shaving, washing, and drying the affected area, the solution was applied daily with a rag or brush, and the pustules squeezed out as they appeared.

In very limited cases after shaving, a dressing of collodion and iodoform to the parts has been used with the object of asphyxiating the parasites, and success has been claimed in occasional cases. Injections into the dermis of 1 per cent carbolic acid around the lesions have been tried. Gmeinin recommends after clipping, washing, and drying the daily application of

Ol. Carui
Alcohol āā 1 part.
Ol. Ricini 15 parts.

and a weekly bath in a 1 per cent solution of potassium sulphide. A change of dressing is often beneficial.

Mange in the Cat

1. **Notœdric mange,** commonly called sarcoptic mange. This disease is due to the *Notœdres cati* var. *cati* (*Sarcoptes minor*) which was noted first by Gohier in 1813 and later described by Hering. The female is 210 μ long by 150 μ broad, while the male is 140 μ long by 100 μ broad.

The disease affects cats of various ages. It is rapidly transmitted from one animal to another. Sometimes it affects the dog, occasionally the horse and man.

Symptoms.—This disease is found most commonly on the head, sometimes the neck, rarely the paws. There is much itching. The cat often scratches; shakes and rubs the head. Frequently it injures the ears with its claws, causing abrasions, scratches, and ulcers. The disease often begins about the ears and forehead. Little nodules appear. Epidermic scales are heaped up, become agglutinated together, and form thick crusts. The fur is cast off in patches. The whole of the head and portions of the neck ultimately become involved, denuded of fur, and covered with thick crusts up to ⅜ inch thick, very firm and adherent, wrinkled and cracked. The eyelids become swollen, inturned, and conjunctivitis is set up. The animal has a most repulsive appearance, loses condition, becomes feeble and emaciated. If the case is neglected, the cat may die of the disease in four to six months.

The diagnosis is easy. The parasites, as a rule, are very numerous in scrapings of the crusts. The disease may thus be easily distinguished from lice or dry eczema.

Treatment is on the same lines as for sarcoptic mange in the dog. The animal must be isolated. The fur is clipped off from head and neck. The crusts scraped and washed off, and then sulphur ointment or liniment used as in the dog.

In advanced cases a cure is not easily effected, and it may be advisable to destroy the animal.

2. **Otodectic Mange or Symbiotic Mange in the Cat.—(Otacariasis, Parasitic Canker of the Ear).**—This disease is due to *Otodectes cynotis* var. *cati.* The parasite is slightly smaller than the variety found in the ear of the dog. It was observed first by Huber in 1860, and later studied by Megnin. It has occasionally been transferred to the dog. The disease is highly contagious, and is very common. It may pass rapidly through a cattery, and is most frequent in Persian cats.

The symptoms and treatment are as for otodectic mange in the dog. Nervous symptoms are not common.

In the **Rabbit** notœdric mange similar to that in the cat has been noted, and otacariasis due to *Psoroptes communis* var. *cuniculi* is quite common, and is similar to that seen in the goat.

J. F. C.

Mange or Scabies in Poultry

1. **Leg Mange or Scaly Leg—Scabies of the Legs.**—This disease is due to *Cnemidocoptes mutans* (Robin and Lanquetin, 1859).

The ovigerous female measures about 420 μ

long by 350 μ broad, while the male is much smaller, being 195 μ long by 125 μ broad.

The disease has been recognized for a long time, but the cause was only demonstrated in 1859 by Robin and Lanquetin. It is observed not only in fowls, but also occasionally in pheasants, turkeys, and partridges.

It is transmitted by contact, especially when poultry are crowded together. It is only slowly contagious, but if the affection is neglected it may spread through the whole of the members of a flock.

Symptoms.—The disease is confined to the limbs. It is accompanied by a certain amount of itching; the fowl frequently scratches or pecks the limbs. The tarsi and the upper parts of the toes are particularly involved. The scales have lost their smooth, regular arrangement. They appear rough and lumpy, and joined together by white powdery crusts. If these are carefully examined, little openings may be found which may be followed by means of a needle to the ovigerous female cnemidocoptes which have burrowed under the scales. These crusts are very adherent. When they are raised up, a red, bleeding surface is exposed. If the inner surface of a crust be examined with a lens, numbers of the female parasite will be detected under a thin, clear pellicle at the end of the little tracts which they have made. These parasites are quite immobile. The crusts are often crumbly, and appear quite spongy. Under the superficial crusts may be found the male and other stages of the cnemidocoptes, which are very active. The disease is very slow in its course, and may continue for six or twelve months. Sometimes the bird becomes lame, unable to perch. Rarely arthritis of one of the joints may be caused, and even necrosis of one of the toes result. The fowl loses condition, ceases to lay, and in advanced cases to feed. Death occasionally results.

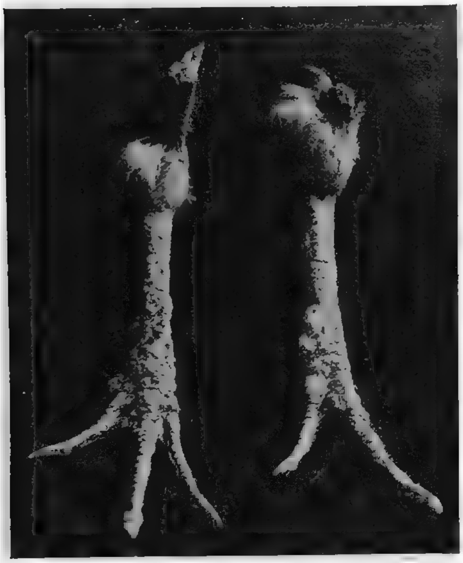


FIG. 128.—Scaly leg in a fowl.

The diagnosis is easily made from the appearance of the limbs and the identification of the parasite after raising up one of the crusts.

Treatment.—The affected animals should be segregated, and the floor and perches which they have used should be disinfected. The limbs should be thoroughly washed with soap and water to remove the crusts, then dried, and a parasiticide dressing applied. Any of the following are useful: sulphur ointment, lime and sulphur dip, kerosene emulsion as used in sarcoptic mange in the horse, or petrol 1 part, rape oil 5 to 10 parts. Two or three dressings at intervals of a few days if thoroughly applied will be sufficient to get rid of the disease.

2. **Depluming Scabies (Feather Rot).**—This disease is met with in chickens and pigeons. It is due to a variety of *Cnemidocoptes lævis*, recognized first by Railliet in 1885 in pigeons and in 1886 in poultry. The parasite is similar to the *Cnemidocoptes mutans*, and the variety found in the pigeons is slightly smaller than that observed in the fowl.

The disease appears to be highly contagious in certain outbreaks described.

Symptoms.—This form of scabies affects the bodies of the birds and commences about the base of the tail or the thighs. Then it extends over the breast, back, and wings in patches. Scales accumulate round the base of the feathers, and the latter become easily detached. The bird often pecks the feathers out. Under the scales and even inside the quills the parasite may be detected. As a result of the loss of feathers bare patches are left and the flying powers of the pigeons are much depreciated. Rarely is there much loss of condition.

The loss of feathers is most common in the spring and summer.

Treatment is on the same lines as in the previous disease in birds. Acaricide applications are rubbed into the parts affected. Sulphur powders or lotions are sometimes used, and their use continued at short intervals for some time.

J. F. C.

THE TREATMENT OF MANGE AND ALLIED SKIN DISEASES IN HORSES BY EXPOSURE TO SULPHUROUS ACID GAS

During the early part of 1917 my attention was drawn to a booklet written by M. E. Lépinay, Vétérinaire aide-major in the French Army, on the above-named method of treating skin diseases, and being struck by the simplicity of the treatment and the ease with which it could be applied " in the Field," I decided to give it a trial, and consequently erected a small chamber capable of accommodating four animals.

This chamber consisted of a wooden erection about 12 ft. 6 in. long by 7 ft. deep, 5 ft. 9 in. high at the back and 7 ft. 3 in. high at the front; it was divided into four stalls by open partitions about 3 ft. 6 in. high. There was also a window at either end, both of which were made to open, so as to allow of the escape of the gas when the treatment was completed but before the collarettes were removed, and the whole was made gas-tight by a paper lining.

The front consisted of four doors, each being divided into two, an upper and a lower, the upper one having an opening 18 in. by 2 ft. 4 in. to hold the framework carrying the collarette; these doors and frameworks were made gas-tight by felt padding.

The sketches on the following page will give some idea as to the construction of the chamber:

The collarettes were made tapering, the posterior or larger end being securely fixed to the wooden framework and the smaller or anterior end being adjustable round the animal's head by means of a running cord; this was placed in front of the ears and above the eyes; care must be taken in adjusting this so as to prevent the escape of gas as much as possible, as the gas quickly causes irritation to the eyes. On experimenting, we found that a piece of blanket, each side being rolled towards the centre—thus forming a groove with a roll on either side—if placed around the head and the collarette carefully placed in this groove, the escape of gas was effectually controlled.

These collarettes gave us some little trouble, as the gas quickly rotted the different materials of which they were made; anything of a vegetable nature was very quickly affected, but I think some woollen material, which will not allow the gas to pass through it, would be found suitable, though I have not been able to give this a trial up to the present.

The generating apparatus consisted of an improvised iron box, inlet and outlet pipes which conducted the gas to and from the chamber, and a fan from an ordinary field forge, this being used to keep the gas circulating. The SO_2 was generated in the box and the fan kept working the whole time so that the gas was being continually fanned into the chamber and exhausted from it through the outlet pipe. It was quite simple and worked very satisfactorily, but, of course, a small engine to drive the fan would be a big advantage, as it is tiring and a waste of time for a man to keep turning the handle for two hours at a time.

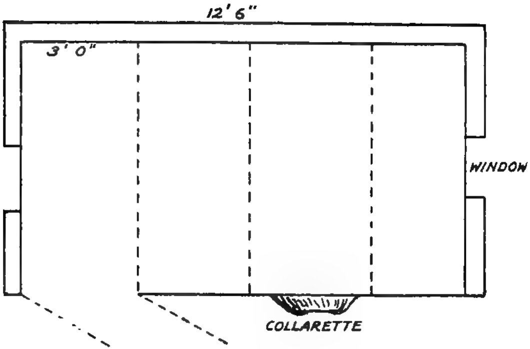
In the booklet referred to it was advised that the animals should be clipped and washed with soda before the treatment was given, but owing to the difficulty in obtaining the soda and the time which it involved, this was discontinued after a time, apparently without detracting from the results, but I think it is advisable to carry out this if possible.

Everything being ready, the animals were backed into the chamber and the collarettes carefully adjusted after the blanket-roll had been placed around the head, all doors and windows securely closed, a netful of hay was hung up in front of each animal to keep him amused and quiet, and the treatment was commenced.

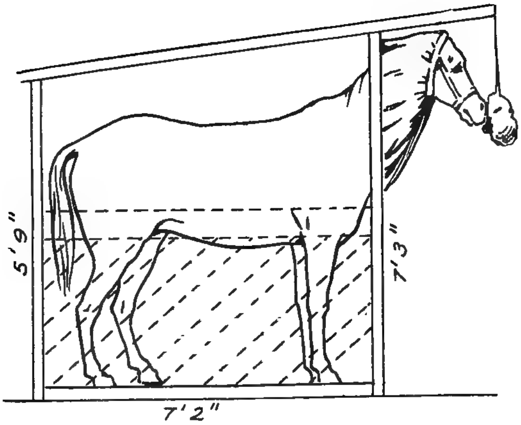
Occasionally a little difficulty was experienced in backing an animal into the chamber, but this was the only difficulty—if it can be called one—which was encountered; when the treatment was commenced the animals appeared to appreciate the warmth and enjoy it and stood quite quietly the whole time. One accident occurred through an animal slipping up and drawing his head into the chamber, but this was guarded against in the future by arranging a movable cross-piece behind each animal so that it could be fitted close to the quarters and thus prevent the animal getting back in the stall; this is the only accident which occurred during the whole time the chamber was in use.

From experiments made it was found to take about fifteen minutes to obtain a 4 per cent concentration in the chamber, so that the two hours' treatment did not commence until the fifteen minutes had elapsed, that is to say, the animals were really two and a quarter hours in the chamber. At the conclusion of the treatment, *i.e.* two hours' exposure to a 4 per cent concentration of SO_2 gas, the two end windows were opened to allow the gas to escape; when clear, the collarettes were removed, the doors opened, and the animals led out. If there was any affection around the head, a little sulphur ointment was applied to the parts, but no other treatment was accorded, although good grooming, exercise, and general care of the animal was insisted on. The first few cases were washed on the third day again after the treatment, but, as before, this was stopped after the first few cases.

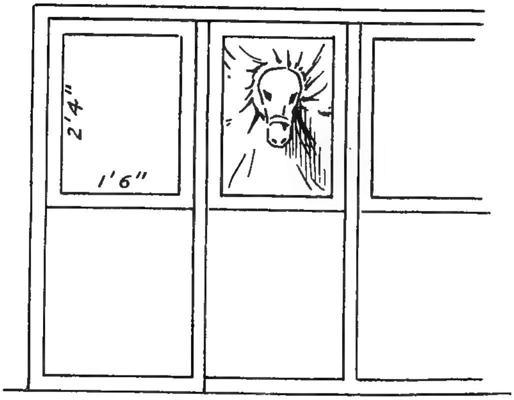
The following remarks concern 354 animals which were treated by this method in four months, September to December. Unfortunately, owing to the many moves of the different units which took place it was found impossible to follow up many of the cases individually, and for this reason definite figures, beyond the number treated, cannot be given in the first report on 303 cases. All the animals sent in for treatment were suffering from various forms of skin irritation, which was acute and persistent. Five were positive cases of sarcoptic mange, and the



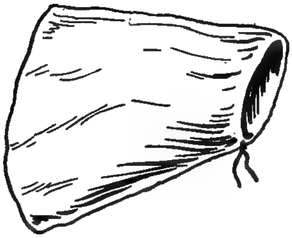
PLAN



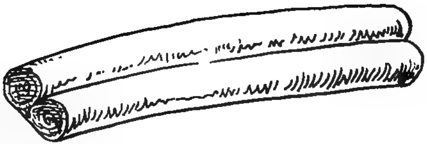
SIDE VIEW



FRONT



COLLARETTE



BLANKET ROLL PLACED AROUND HEAD TO RECEIVE NOOSE OF COLLARETTE majority (75 per cent) of the remainder were diagnosed by the Veterinary Officers sending them in as "suspected mange," and although we were often unable to isolate the parasite, I am convinced many more were true mange cases; but any one knowing the conditions which existed in the forward area—and I might mention that my gas chamber was within eight miles of the front line—would, I think, forgive us for not always finding the parasite, and allow us to draw our own conclusions from the macroscopic appearances.

It was found that after one application the irritation generally remained for some ten to fourteen days, although gradually subsiding, but at the end of three weeks the animal showed a really wonderful improvement; practically a new coat had grown and the skin was perfectly clean and supple, whereas before the treatment was given it was harsh, scurfy, and in many cases corrugated and quite denuded of hair.

Owing to this delay in the arrest of the irritation our first treatment of a positive case of mange was a failure. The animal was subjected to one exposure of two hours and no further treatment was applied. At the end of a fortnight it was found that the irritation was not so acute and that the skin had improved in appearance, but that a cure had not been effected. The animal was therefore allowed to remain for a week without being touched, and was then subjected to an exposure of two hours on three successive days. This took place on 16th October, and at the end of three weeks the skin was clean, irritation entirely ceased, and the animal was considered cured; this case was kept under observation until it moved out of the area two months later, and no further symptoms of mange developed, it being in perfect condition when it left. I subsequently received a report from the Veterinary Sergeant who had charge of the animal; it is dated 23rd January; in it he says: "I may mention that the grey cob is in fine condition, quite as good as when you saw it last."

Four other positive mange cases were treated in a similar manner, *i.e.* two hours' exposure on three successive days; three were discharged cured, but the fourth was evacuated by the Veterinary Officer as the unit was moving, and as the animal was debilitated he did not consider it fit to march. I was unfortunately not aware that this had happened until after the animal had gone.

Of the remaining 298 animals which were treated, not one of them developed mange by the time we left the area (the end of December) although no other treatment than one exposure to SO_2 was given, with a few exceptions, when the exposure was repeated a second time after an interval of ten days to a fortnight, and the application of a little sulphur ointment around the head as already mentioned; and as far as I am able to ascertain not one of these animals was subsequently evacuated for any form of skin trouble, whereas quite 75 per cent of the cases would have been evacuated as suspected mange had not this treatment been carried out. I think I am also right in stating that it was found the majority of cases sent to the Base Hospitals as suspected mange were proved to be true mange cases or due to the forage acari. Another interesting fact noted is that the reduction in the number of cases of mange and suspected mange reported each week during this period amongst the animals under my administration fell from seventy-six in the first week to five in the last week; to my mind that is very satisfactory evidence as to the value of this treatment.

From experience, I consider the results obtained justify one in concluding that this is a most promising treatment for skin affections, whether it be mange or pruritus. I am quite convinced that sarcoptic mange can be cured easily by this treatment; the method is cheap, clean, easily worked by one intelligent man, it takes a very short time in comparison with other methods, and the results are quickly seen.

The improvement in the general condition of the animal's skin is appreciated almost immediately, and it is really remarkable. If I had not had the experience myself, I could not have believed it possible; I have known officers dispute as to the animal being the same one which they had seen three weeks previously; from harsh, staring coats and dry skins, often corrugated and denuded of hair, they have perfect coats like satin and beautifully clean, healthy skins to such a degree as to give rise to the most agreeable criticisms.

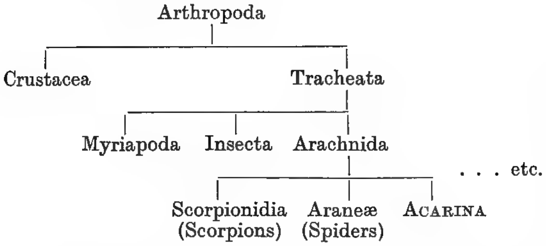
Of course it is appreciated that by this method the same number of animals cannot be treated in a specified time as is the case when using a dip, but from my present experience I consider the SO_2 treatment, as I have endeavoured to describe it, is as effectual, quicker, and infinitely cheaper both in the cost of material and in man power; in fact in every way.

M. Lépinay states that mange can be cured by this method by one exposure of two hours, but that was not my experience; on the other hand, it must be remembered that our apparatus was quite primitive and roughly made, which may have made a considerable difference. I was hoping to have been able to carry out some further experiments, but unfortunately we had to dismantle the chamber when we left the area, and although I eventually had another larger one, for twelve animals this time, erected, I much regret to say we had to hand this over hurriedly to the enemy just as it was completed, with the exception of fitting the collarettes, but as he was apparently in somewhat of a hurry to get it we left the finishing to him to carry out. However, I am still hoping to carry out the experiments under more favourable circumstances.

E. M. P.

TICKS

Ticks are members of the Order Acarina, whose position in the animal kingdom is indicated in the following scheme :



The Acarina are usually very minute animals and are commonly known as Mites. Itch Mites (*Sarcoptes*) and Cheese Mites (*Tyroglyphus*) are familiar examples. They are characterized by an absence of segmentation. In all Arachnids the head is fused with the thorax, but in mites there is no "waist," the thorax being fused with the abdomen, which is itself unsegmented. Moreover, there is a characteristic metamorphosis, the egg giving rise to a six-legged "larva," which changes to an eight-legged sexless "nymph," which again changes to the eight-legged male or female "imago." In some cases more than one nymphal stage is recognizable. The Acarina are divided into groups according to the position of their breathing organs, and the ticks, having their breathing orifices somewhat posteriorly situated, are included in the Metastigmata, of which they comprise two families, Argasidæ and Ixodidæ, which, taken together, form the super-family Ixodoidea.

The Ticks or Ixodoidea are, then, comparatively gigantic Acarines of external parasitic habit, living entirely on the blood (or lymph) of terrestrial vertebrates of every kind—mammals, birds, reptiles, and amphibia. The two families Argasidæ and Ixodidæ differ somewhat widely both in structure and manner of life, but both agree in the possession of characteristic mouth-parts, which distinguish them from all other acarines.

Though ordinary segmentation is wanting, and there is no true head, a small portion of the anterior part of the body is movably articulated, and is known as the false head or *capitulum*. It consists of a basal portion (*basis capituli*) produced anteriorly into a piercing apparatus flanked by a pair of more or less mobile palps.

The piercing apparatus has the following structure. The actual cutting organs are the *chelicerœ*, a pair of extrusile jaws each consisting of a shaft and a digit, the digit being capable of lateral motion, and toothed on its outer edge. Ventral to the *chelicerœ* is a rigid *hypostome*, arising from the *basis*, and furnished beneath with a number of recurved teeth, so that, when thrust into the wound cut by the *chelicerœ*, the parasite is firmly anchored. In the Ixodidæ the *chelicerœ* are covered dorsally by a chitinous structure arising from the basis and known as the *sheath*. Both *hypostome* and *sheath* show signs of being double structures though the two halves are fused (Fig. 128).

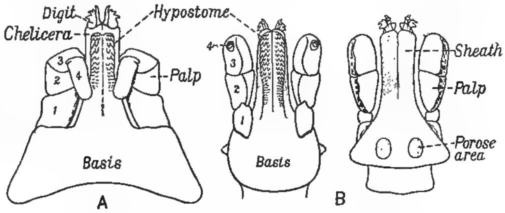


FIG. 129.—Capitulum.

A, capitulum of Argasidæ; B, of Ixodidæ (*Hyalomma*) in ventral and dorsal aspect.

The possession of this characteristic capitulum is common to all ticks, and readily distinguishes them from other acarines with the single exception of the Gekobiidæ, an obscure group of minute Prostigmatic Mites parasitic on reptiles. They are allied to the Harvest Mites (Trombidiidæ), and have the same tendency to a scarlet coloration. Their similarity in habit to the true ticks has brought about a modification of their mouth-parts on somewhat similar lines.

FAMILY I. ARGASIDÆ

The Argasidæ are ticks with leathery integument, no portion being specially chitinized to form a *scutum* or shield. The sexes differ very little. The capitulum (in nymphs and adults) is inferior, and invisible dorsally, and the palps are leg-like. The spiracles are small and usually anterior to the fourth leg. The pad (*pulvillum*) which characterizes the foot of the Ixodidæ is rudimentary or absent.

In habits the Argasidæ somewhat resemble the bed-bug (*Cimex lectularius*). Their chief hosts are man, birds, and bats, and they are as a rule nocturnal, feeding moderately and at frequent intervals. When fasting they are flat and readily creep into crevices. When gorged there is no extravagant increase in size, as in female Ixodidæ, but they become thicker. They live for several years.

The bite of the Argasidæ is generally painful, and massive infestation may have serious results. The transmission of definite blood parasites has been proved in the case of three species—*Argas persicus*, *A. reflexus*, and *Ornithodorus moubata*. Two genera are recognized, *Argas* and *Ornithodorus*, but the characteristics which distinguish them are not of high importance.

GENUS 1, **Argas.**—*Body with a distinct margin, of different texture to the rest of the integument, and not entirely obliterated on gorging. Eyes absent.*

There are only six well-established species. *A. vespertilionis*, remarkable for its circular shape, is parasitic on bats and has been taken in England, France, and (a variety) at the Cape. It has been called the Blyborough Tick. *A. æqualis* and *A. transgariepinus* rest on very few specimens taken in Africa, and little is known of them. *A. persicus*, *A. reflexus*, and *A. brumpti* have more or less economic importance and require fuller treatment.

A. persicus (syn. *miniatus*) is known by several popular names: Fowl Tick (America, Australia, Africa), Adobe Tick (America), Wandluis or Tampan (S. Africa), Miana (or Miane) Bug (Persia), etc. It is cosmopolitan in fairly warm climates. The margin, which in other species is striate, is formed of quadrangular cells.

Hosts.—Birds (especially domestic fowls) and man.

Life-history.—Eggs in batches of 20-100 are laid, about a week after feeding, in crevices (in fowl-houses, tree-bark, etc.) and hatch in about three weeks. If unfed the larvæ die within eight weeks, but if they find a host they remain attached five to ten days and drop off gorged. In about a week the first nymphal form emerges. Henceforward the feeding is rapid, occupying 1½ to 2 hours. In about two weeks another moult gives rise to the second nymphal form. These feed and give rise to adults after several weeks. Adults feed about once a month during hot weather, the females laying eggs after each meal.

Relation to Fowls.—*A. persicus* injures fowls by (*a*) massive infestation; (*b*) communicating the parasite *Spirochæta gallinarum.*

(*a*) Massive infestation alone is quite capable of causing great mortality in fowls. The ticks crowd most thickly under the wings, but may cover the whole skin.

(*b*) Spirochætosis may be fatal to a whole brood in a few days. The first symptoms are diarrhœa, loss of appetite, somnolence, ruffled feathers, and pale comb. The birds cease to perch, lie down, and die in convulsions. Sometimes the course of the disease is less rapid; the legs are paralysed, then the wings. Emaciation and death occur in eight to fifteen days. Recovery is rare after paralytic symptoms have set in. The spleen is much enlarged and the liver swollen, with fatty degeneration. The blood is fluid and dark, and abounds with spirochætes until shortly before death.

Relation to Man.—The bite of this tick is very painful, and has frequently been stated to have fatal results, but accounts are probably exaggerated. There is no proof of its ability to communicate relapsing fever.

Destruction of Ticks.—Birds may be cleared of ticks by dressings of petroleum or turpentine, but the thorough cleansing of fowl-houses is the principal measure. Old roosts and nests should be burnt, and the walls of the fowl-house brushed with hot coal-tar. Kerosene emulsion, lime and sublimate, and "creozozone" are advocated as sprays for hencoops and poultry-yard. Roosts should have no bark, and should be suspended by wires so as not to touch the walls.

A. reflexus (syn. *marginatus*), the Pigeon Tick, is only found in Europe and North Africa. Its only known habitat in England is Canterbury Cathedral, but it occurs in France, Germany, Italy, Russia, Rumania, and Algiers in pigeon-coops.

Hosts.—Pigeons and man. In one case it was taken from a horse.

Life-history.—Probably much like that of *A. persicus*, but this tick shows remarkable longevity. Berlese kept an unfed specimen alive for six years in a corked bottle. Unfed larvæ live six to eight months.

Relation to Disease.—Though experiments have proved that this tick is capable of communicating *Spirochæta gallinarum*, its normal injury to pigeons appears to be by massive infestation, young birds being the principal sufferers. Human beings sleeping near pigeon-cotes are often attacked. Pain, itching, and sometimes erythematous eruptions ensue. An instance cited by Tonnel seems to show that *A. reflexus* can transmit pyrogenic bacteria.

Treatment.—As for *A. persicus.*

A. brumpti.—Brumpt found this tick at Ogden in Somaliland, in dusty sheltered places beneath overhanging rocks, into which opened porcupine burrows. The ticks hid in the dust by day and attacked Brumpt's party at night, causing ecchymosis and pruritus which lasted many days. After seven years the skin was still indurated, and there was occasional pruritus in the case of a few of the bites.

GENUS 2, **Ornithodorus.**—*No distinct margin. Body often narrowed in front to a hood-like projection over the capitulum. Eyes present or absent.*

About twelve species of *Ornithodorus* have been described, but of some, which have been taken on the ground, neither the hosts nor the habits are known; but *O. coriaceus* (Paraguay, Mexico, California), which inflicts a painful bite on man; *O. tholozani* (Persia), on fowls and camels; *O. lahorensis* (Lahore), on *Ovis aries*; and *O. savignyi* (widely spread over Africa, but more common in the north and east), on fowls, dog, goat, cattle, and pig, are species which appear to be fairly innocuous. *O. savignyi* has been experimentally proved capable of conveying the spirochæte of relapsing fever, and may perhaps be the natural carrier in Somaliland and regions beyond the northern range of *O. moubata*. *O. turicata* (Texas, Florida), which has been found on cattle, pig, llama, and various wild animals, and *O. talaje* (Southern United States and Central America), which infests old houses and attacks the hands and ears of man at night, are two species with an exceedingly evil reputation locally for the severe effect of their bite on man, which causes intolerable itching, pain, ecchymosis, sometimes followed by abscesses, erysipelatoid dermatitis, etc. *O. megnini* (Mexico and south-west parts of the United States), the "spinose ear tick," infests the ears of the horse, ass, and ox, and frequently enters the human ear.

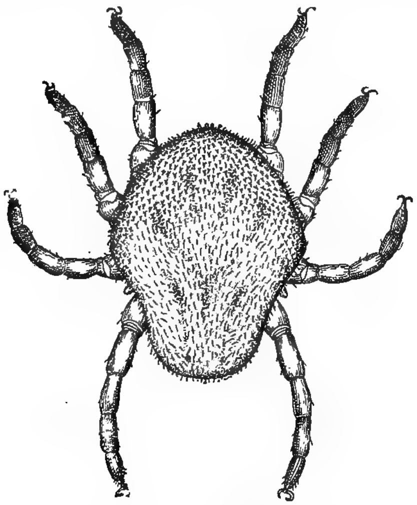


FIG. 130.—Nymph of *O. megnini* (unfed), magnified about 10 times. Dorsal aspect.

The *life-history* is remarkable. The larvæ bury themselves in the folds of the skin of the ear, and gorge. In about five days they moult, still in the ear, and the nymphs, which are very spinose and differ greatly from the adult, continue feeding for a long time—one to three months. They then leave the ear and give rise to adults which, *without further feeding*, pair and oviposit.

There is much difference of opinion as to the importance of this parasite, some attributing to it sickness and even death among cattle, while others regard it as merely causing annoyance. The ears may be cleared by the application of any bland oil, or by the introduction of a pledget of cotton-wool with a little chloroform. The only known species of *Ornithodorus* which is distinctly pathogenic is *O. moubata*, a species closely resembling *O. savignyi* but without eyes, occurring only in Africa, where it is widely distributed, and greatly feared by the natives under the names "moubata," "bibo," "papaze," etc. It is the carrier of *Spirochæta duttoni*, the cause of relapsing fever in man. It also attacks domesticated animals.

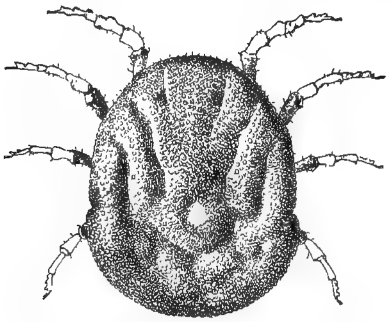


FIG. 131.—*O. moubata*, × 4. Dorsal aspect

Life-history.—*O. moubata* inhabits native huts and rest-houses, hiding by day and feeding by night. It is often transported in beds and matting. The females burrow in the sand and lay batches of 10-20 eggs at intervals of one to two weeks. The maximum number observed was 150. The larva does not feed, but while still within the egg-shell develops into a first-stage nymph, which emerges ten to fifteen days after oviposition. There are three or four nymphal stages before the adults appear. Nymphs and adults attack the host, taking 10 to 120 minutes to gorge.

Relation to Disease—*O. moubata* communicates *Spirochæta duttoni*, which is a parasite both of man and of the tick, for a female which has imbibed blood containing the spirochæte conveys the parasite to its offspring, which is infective up to the third generation at least. Its excreta are also infective. The main symptoms of relapsing fever are "headache, vomiting, abdominal pain and purging with severe fever, a pulse of 90·120, dry hot skin, congested eyes, and shortness of breath" (Nuttall). There is a fall of temperature after about two days, but there are usually five or six relapses.

Spirochætosis in fowls can be transmitted by this tick, and it has been suspected that it is capable of transmitting Filariasis to man.

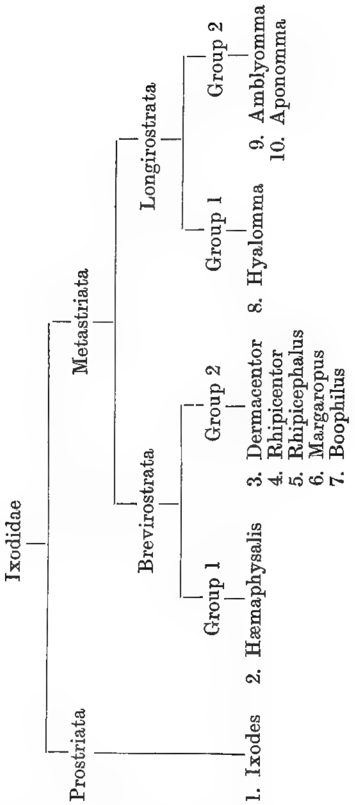
Prevention.—Infested huts should be burnt, and native rest-houses shunned. Sleeping in hammocks and under mosquito nets mitigates the danger of infection.

FAMILY II. IXODIDÆ

This family comprises the great bulk of the ticks. The capitulum is always terminal, and the fourth or final article of the palp is so reduced in size as generally to form a mere tubercle beneath article 3. A *scutum* or shield is always present. The sexes are very distinct, the scutum covering the whole dorsum of the male but only the anterior portion of that of the female, nymph, and larva. The males feed little or not at all. The other stages imbibe large quantities of blood and increase immensely in size, so that the scutum becomes a mere patch on the anterior part of their greatly distended bodies. They take several days to gorge themselves, and each stage feeds but once. The replete female oviposits on the ground and then dies. Oviposition may occupy several weeks, and the eggs are generally very numerous, *Amblyomma hebræum*, for example, having been known to lay 18,500. The six-legged larvæ which hatch out climb neighbouring herbage and await the passing of a host. If they are fortunate they gorge, taking some days over the process, and then generally drop to the ground, where after a quiescent interval they moult, giving rise to eight-legged but sexless nymphs. These behave similarly but take a longer time to gorge, often remaining some weeks on the host. On dropping off they again remain quiescent for a time before giving rise to the adult ticks, which for a third and last time seek a host. Pairing may take place either on the ground or on the final host. Such ticks (the majority) are known as "three-host ticks," three different animals being required for the completion of their life-history. There are, however, "two-host" and "one-host" ticks. In the former case (*e.g. Rhipicephalus evertsi*) the replete larva changes to a nymph on the first host, which is continuously attacked by both larva and nymph. In the second (*e.g.* the genus *Boophilus*) the tick remains on the first host through all its stages until the replete female drops off to oviposit.

Under the most favourable conditions months are usually occupied in the life-cycle from egg to egg, but the period may be greatly extended. Cold retards development, and there may obviously be indefinite delay in finding suitable hosts in the case of three-host ticks. All stages are very tenacious of life in a fasting condition.

Ten genera of Ixodidæ are usually recognized, and the following scheme indicates their affinities.



Ixodes differs more from the other genera than they do from each other, and is set apart under the name Prostriata because the anal groove passes round in *front* of the anus. The other genera, in which the anal groove (unless obsolete as in *Boophilus*) passes *behind* the anus, fall naturally into two divisions, in one of which (Brevirostrata) the piercing apparatus is short, in the other (Longirostrata) long. Of the Brevirostrata *Hæmaphysalis* alone is eyeless, and its peculiar capitulum and the absence of male armature characteristic of the rest of the group set it somewhat apart.

Of the Longirostrata the genus *Hyalomma* only contains very few species, but one (*H. ægyptium*) is exceedingly common and widely distributed. *Amblyomma* is a large genus, and contains at least one highly important pathogenic species, *A. hebræum*, which communicates "heart-water" to cattle. *Aponomma* differs little from *Amblyomma* except in being eyeless, and ought probably to be regarded as a subgenus. The fact that its hosts are almost always reptiles deprives it of any economic significance.

Many ticks are "ornate," their scuta being variegated by white or coloured or metallic markings. This is usually the case in *Dermacentor*, *Amblyomma*, *Aponomma*, and is also occasionally to be found in *Rhipicephalus*. An "ornate" tick, then, must belong to one of these genera. Ticks not so adorned are termed inornate.

The points of structure to which attention should be specially directed in attempting to identify a tick are the following: (1) the *capitulum*: the shape of the basis and of the palps, and the form and dentition of the hypostome; (2) the *scutum*: its colour, shape, punctations, grooves, and the presence or absence of eyes, and, in the male, its posterior folds or "festoons"; (3) the *ventral aspect*, the position of the sexual orifice and anus, the spiracle and (if a male) the nature of such chitinized plates or shields as may be present; (4) the *legs*, particularly the first joints (coxæ) and the terminal joints (tarsi).

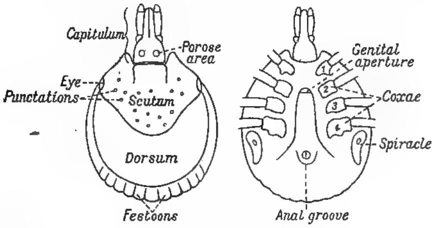


Fig. 132.—Diagrammatic dorsal and ventral view of a female Ixodid tick (*Amblyomma*).

The different genera of the Ixodidæ will now be discussed in the order indicated above. The forms which infest man and domesticated animals will especially concern us, and particular attention will be directed to those which have been proved to be pathogenic.

Prostriata

Genus 1, Ixodes.—*Inornate ticks without eyes or festoons; article 2 of the palps narrow at the base; spiracles round or oval; sexual dimorphism pronounced, the venter of the male being covered by chitinized plates which do not project. Anal groove in front of anus* (see Fig. 133).

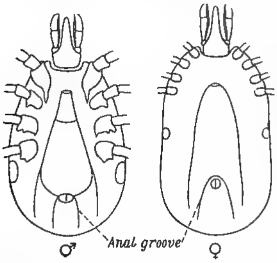


Fig. 133.—Diagrammatic ventral view of ♂ and ♀ Ixodes. Note the prostriate anal groove.

There are about fifty known species of *Ixodes*. Some exclusively attack bats and other birds, but the majority have been taken from mammals, and do not at all confine themselves to a particular host, though they may show a preference for a group of mammals of somewhat similar habits. Of those parasitic on birds those which affect marine birds (*e.g. I. putus*) may have a very wide distribution In the case of many species the males are unknown, and in few cases where they are known they have never been found on the host. Nuttall has shown that this fact is correlated with the habits of the host If it be a wandering animal, like the sheep, male and female ticks are found on it; if there be a nest or tolerably fixed lair the male tick remains in that retreat and usually has its piercing apparatus feebly developed.

The bite of *Ixodes* is not generally particularly severe, though *I. putus* is said to inflict a painful wound, and *I. ricinus* (*vide infra*) may induce serious symptoms. *I. pilosus* (all domestic and several wild mammals, Cape Colony) is suspected of producing "paralysis" in sheep, and *I. rubicundus* (sheep, Eastern Cape Colony) has been accused of being the cause of an obscure disease characterized by anæmia, but the only species which has been definitely proved to be pathogenic is *Ixodes ricinus*, the European "sheep tick." (There is a long spine on coxa I. and the tarsi are tapering.) It attacks sheep, dogs, cattle, and a large number of (generally wandering) wild mammals. This species, or some variety of it, extends throughout Europe and North America and is also recorded from North Africa and Transcaucasia. It is a three-host tick.

Effect of the Bite.—Psoriasis-like eruptions and phlegmonous inflammation are frequent results. When the tick has been roughly removed, leaving the capitulum behind, grave effects have sometimes followed. When the bite was at the back of the head, swelling, headache, stiffness and cramp in the muscles of one side, partial loss of memory, polyuria, dilatation of the pupils, etc. resulted (Johannessen). Gangrenous inflammation in sheep, and abscess, œdema, lymphangitis, etc., in man, have followed the bite of this tick (Mauvezin)

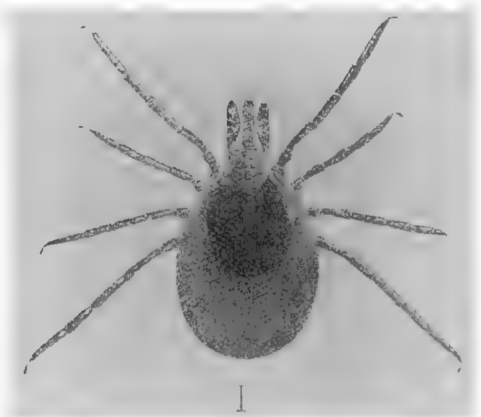


Fig. 134.—*I. ricinus*, ♀, unfed, much enlarged. (After Wheler.)

There are several cases recorded in which *I. ricinus* has penetrated the skin of man and various animals and entirely embedded itself, with no sign of external lesion.

Relation to Disease.—*I. ricinus* has been suspected of transmitting canine piroplasmosis, and there is good reason to believe that it is the cause of a disease in sheep (Chorea paralytica ovis) prevalent in Scotland and the north of England, where it is known as "louping ill." It has been shown (Kossel and others) to be capable of transmitting redwater (bovine piroplasmosis) to cattle, a disease characterized by fever, progressive emaciation, anæmia, and urine coloured a deep red by hæmoglobin. An intravenous injection of 200 c.c. of a 1·5 per cent aqueous solution of trypanblue is wonderfully successful in curing the disease, but the animals remain infective (Nuttall and Hadwen). See also *Boophilus* and *H. cinnabarina* var. *punctata.*

Another very common English tick is *I. hexagonus* (short spine on coxa I., and humped tarsi). It occurs on the dog and sheep, but especially on the hedgehog and wild carnivora.

METASTRIATA—BREVIROSTRATA

GENUS 2, **Hæmaphysalis.**—*Inornate ticks with festoons but without eyes. Characteristic capitulum, the palps being conical and broadest at the base of article 2, which projects laterally beyond the basis. Sexual dimorphism slight, the male having no ventral plates or shields, though it may present special spines on the palps and the coxæ.*

These are usually small ticks, yellow or brown in colour, and the general integument is rather highly chitinized, so that the scutum and other hard parts stand out with less than the usual relief. The second joint (trochanter) of leg 1 always has a dorsal backwardly projecting spur (see Fig. 135), and a corresponding ventral spur may be present.

About fifty species are known, and most of them are Asiatic. A few species are peculiar to Madagascar and six or seven occur in Africa. Probably a better knowledge of the Australian fauna would increase the scanty number of species so far found there, but the rest of the world is poor in representatives of this genus. Most attack mammals, but a few are bird parasites. Some of the species which normally attack mammals have on occasion been taken on reptiles.

Few species of *Hæmaphysalis* have any economic importance, and two only—*H. leachi* and *H. cinnabarina* var. *punctata*—have been proved to be definitely pathogenic, but it must be recorded that *H. bispinosa* occurs plentifully on domesticated animals in India, and that *H. inermis*, a very aberrant form which differs from all other Ixodidæ in the rapid manner in which its nymphs and larvæ feed, attacks deer in France and cattle in Transcaucasia.

H. cinnabarina var. *punctata* (often called *H. punctata*) is distributed over Europe and the adjoining parts of Asia and Africa. It has been taken from a great variety of hosts, including domesticated animals. In England it occurs in South Wales and in Kent, where it attacks sheep. On the Continent it attacks cattle and has been proved able to communicate to them *Piroplasma divergens*, the cause of European "red-water," so that while *Ixodes ricinus* is without doubt the main instrument in transmitting that disease it may be aided by *H. cinnabarina* var. *punctata*. *H. cinnabarina*, the type variety, occurs in North America.

H. leachi is the African dog-tick. It has been taken on a great number of wild animals, chiefly carnivora, and though undoubtedly of African origin, has spread to various parts of the world, probably through its partiality for *Canis familiaris*, and is recorded from many parts of Asia and from Australia. It transmits the protozoan parasite *Piroplasma canis*, the cause of canine piroplasmosis or malignant jaundice in dogs, an exceedingly deadly disease, whose principal symptoms are high fever, hæmoglobinuria, jaundice, anæmia and a watery condition of the blood. It is cured by an intravenous or subcutaneous injection of 10 c.c. of a 1½ per cent aqueous solution of trypanblue administered at an early stage of the disease (Nuttall and Hadwen). It is remarkable that in the case of this tick infested larvæ and nymphs are not infective, whereas the adults are. The progeny of an infected female harbour the parasite but cannot transmit it till they become adult. (See also *Rhipicephalus sanguineus.*)

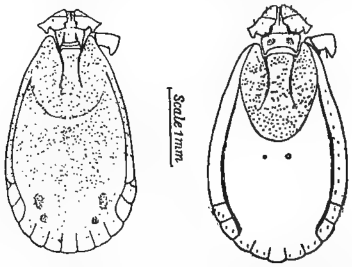


FIG. 135.—*H. leachi*; dorsal view of ♂ and unfed ♀. One trochanter of leg 1 is given to show the spur. (Nuttall and Warburton.)

The development of *H. leachi* is rather rapid for an Ixodid tick, and the whole life-cycle may, under favourable conditions, occupy about one hundred and twenty days. There are probably two broods within the year.

GENUS 3, **Dermacentor.**—*Usually ornate, with eyes and festoons. Basis capituli rectangular;*

palps rather massive. Male without ventral plates or shields, but with enlarged coxa IV. Coxa I. with two strong spurs.

Only about fifteen species of *Dermacentor* are known, and most of these are American. There are two African species (*D. rhinocerotis* and *D. circumguttatus*) and one Asian (*D. auratus*) of no economic importance, and one (*D. reticulatus*) which occurs on domestic animals (dog, horse, sheep, cattle) throughout Europe and part of Asia, but some of the American species are distinctly injurious to dogs, horses, etc., and one (*D. venustus*) is the only tick which shares with *O. moubata* the distinction of being able to transmit a serious disease to man. It is the carrier of "Rocky Mountain spotted fever."

D. nitens (a very aberrant species, being inornate, and with a spiracle unlike that of any other tick) occurs on horses, mules, etc., in tropical America and the West Indies. The attack is practically confined to the animals' ears unless the parasites are so numerous as to overflow, so to speak, to other parts. This is a one-host tick, and has a rapid development. Larvæ placed in the ear turned to nymphs in about a week and fell off as fully gorged females within a month (Hooker, Bishopp, and Wood). Larvæ unable to find a host generally die in a week or two, and under very favourable conditions seldom survive three months.

Relation to the Horse.—Massive infestation of the ears, together with the filth and decaying matter consequent on the presence of the parasites, often cause extensive suppuration, and facilitate the entrance of the "screw-worm."

Remedial Measures.—This is one of the easiest ticks to control. Kerosene and lard applied to the ears kills the ticks. "The keeping of stock from an infested pasture for four months in summer will probably be sufficient to ensure the starvation of the seed ticks" (Hooker, Bishopp, and Wood).

D. variabilis (often referred to as *D. electus*) is known as the American dog-tick, and occurs on dogs throughout the United States, being especially common east of the Mississippi, but no great injury is attributed to it.

D. occidentalis is very common on live-stock and often attacks man in California and Oregon, where it is known as the "wood-tick." It was formerly confounded with *D. venustus* and thought to transmit spotted fever, but it only injures cattle by massive infestation and man by its painful bite.

D. venustus occurs in the Rocky Mountain region, and is most abundant in Montana, Idaho, and Wyoming. Its immature stages are found on various small mammals, but the adults have only been taken from horses, cattle, deer, mountain goats, and man. Its great importance lies in its ability to transmit to man a serious disease, Rocky Mountain spotted fever, which in its mild form entails a mortality of 5 per cent, and in a specially virulent form, occurring in the Bitter Root valley in Montana, is extremely deadly, the mortality rising to 70 per cent. The obscure affection known as "tick paralysis" is also communicated by this species. The tick is only found on cattle from March 1 to June 1, a period to which cases of spotted fever are restricted. This is a three-host tick. The larvæ, if unfed, die in about two months. The life-cycle usually occupies two years, and the winter is passed in the fasting nymphal and adult conditions.

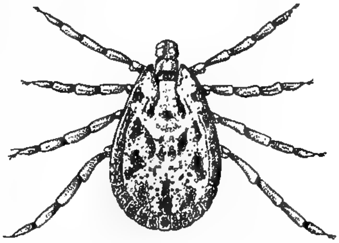


FIG. 136.—*D. venustus*, ♂. × 6. (After R. A. Cooley.)

Destruction of Ticks.—As the adults feed almost exclusively on large domestic animals, dipping, spraying and hand-picking are the most promising measures, but to be effective they must be persevered in for at least two seasons.

GENUS 4, **Rhipicentor.**—Includes only two rare species of African ticks, and need not detain us. It is to some extent intermediate between *Dermacentor* and *Rhipicephalus*.

GENUS 5, **Rhipicephalus.**—*Usually inornate, with eyes and festoons. Basis capituli hexagonal; male with a pair of projecting adanal shields and often with a second pair external to them. Coxa I. with two strong spurs. Spiracle comma-shaped.*

Rhipicephalus is essentially an African genus. *R. sanguineus*, being a dog parasite, is practically cosmopolitan in tropical and subtropical countries, and *R. bursa* has invaded the European countries of the Mediterranean region, but except these only one or two extra-African species are known.

Several of the African forms are pathogenic, and *R. sanguineus*, though of minor importance to *H. leachi* in the country of its origin, is, in India, the principal carrier of canine piroplasmosis.

The determination of species in this genus is particularly difficult because of the large amount of variation observable within each species, while very few species depart widely from the general type. There is evidence that the genus is in a very fluid condition, and many of the so-called species are perhaps in the course of formation and hardly yet definitely established as distinct.

About thirty-five species are recognized.

R. sanguineus, "the brown dog-tick," has been found on many mammals, but has a particular preference for the dog, by which it has apparently been carried to all the warm regions of the world. In Africa its importance as a disease carrier is eclipsed by *H. leachi.* It is a three-host tick, and under optimum conditions it may run through its life-cycle in sixty-three days, but this may be greatly extended by cold weather and inter-stage delays, for this tick is very tenacious of life in a fasting condition.

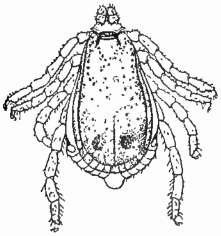


FIG. 137.
R. sanguineus, ♂. ×8.

Relation to Disease.—*R. sanguineus* is the principal carrier, in India, of *Piroplasma canis*, the cause of malignant jaundice in dogs (for symptoms and treatment see *Hæmaphysalis leachi*). The progeny of infested ticks are infective, and do not, as in the case of *H. leachi*, remain innocuous until adult, for they can communicate the disease in the nymphal stage.

R. bursa is recorded from several parts of Africa and from the European countries bordering the Mediterranean. It closely resembles *R. sanguineus*, but is a two-host tick, the change from larva to nymph taking place on the first host. Under favourable conditions its life-cycle occupies one hundred and ten days.

Relation to Disease.—*R. bursa* transmits *Piroplasma ovis* in Italy, Turkey and especially in Rumania, the disease being particularly virulent in the low lands at the mouth of the Danube, where it is known as "Carçeag." There is fever, shivering, loss of appetite, and collapse. Diarrhœa is a frequent symptom, and occasionally there is hæmoglobinuria. Half the infected sheep usually die about the third day after the malady has declared itself (Neveu-Lemaire).

R. appendiculatus occurs on cattle (and many other animals) throughout Central and Southern Africa, and is a three-host tick with a minimum life-period of about one hundred and fifteen days.

Relation to Disease.—This is the principal carrier of *Theileria parva*, the protozoon which causes Rhodesian or East Coast fever in cattle. Ten to seventeen days after infestation there is high fever, and the parasites abound in the blood, increasing till death ensues in about three weeks. In this case cattle which recover are no longer infective. The disease is not transmitted by the tick to its progeny through the egg, but it is passed on from stage to stage, infected larvæ giving rise to infective nymphs, and infected nymphs to infective adults (Nuttall).

R. simus is also a carrier of *Theileria parva*, though of less importance, and *R. capensis* has been experimentally proved capable of transmitting the disease.

R. evertsi (noteworthy for its dark scutum with hemispherical eyes, and its orange-coloured legs) attacks various mammals but especially horses in the Transvaal. It is a two-host tick, the change from larva to nymph taking place on the first host.

Relation to Disease.—*R. evertsi* communicates equine piroplasmosis in the Transvaal, but at the time this discovery was made the two protozoan parasites, *Piroplasma caballi* and *Nuttallia equi*, had not been discriminated, and it is uncertain which of them is in question.

GENUS 6, **Margaropus.**—Comprises a single rare South African species, *M. winthemi*, of no economic importance. The male is remarkable for the extraordinary development of the fourth leg and its unique ventral armature.

American writers as a rule attribute to this genus the ticks which we include in the genus which follows, and the *M. annulatus* of many American publications is the *Boophilus annulatus* considered below.

GENUS 7, **Boophilus.**—*Inornate ticks with eyes but without festoons. Palps very short and transversely ridged dorsally. Female scutum very small and hardly punctate. Male with four sub-equal anal plates. Anal groove obsolete.*

This genus is parasitic on domestic and other animals but especially on cattle in many parts of the world, notably Africa, India, Australia, and the south-eastern part of the United States. Various species have been established, the most widely recognized being *Boophilus* (*Margaropus*) *annulatus* in North America, *B. decoloratus* in Africa and *B. australis* in South America, Africa, Asia, and Australia. Their specific rank is at least doubtful, and is fortunately unimportant, for all the varieties are of similar habit and equally pathogenic. They are small ticks, the unfed females only measuring about 3 mm., and the males 2 mm. The gorged females average about ½ inch in length.

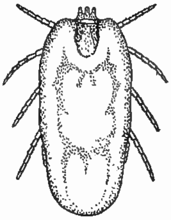


FIG. 138.—*Boophilus*, ♀, partially fed. ×8. (After Birula.)

Life-history.—*Boophilus* is a one-host tick. The female lays a large number of eggs (up to about 5000) on the ground, and the larvæ climb the neighbouring herbage to await a passing host. On this the further changes are passed through. The larva moults and the nymph re-attaches itself and presently gives rise to an adult which gorges and pairs before dropping to the ground, so that all stages are found on the host at the same time, and at any time of the year. Their development in hot weather is rapid, and *B. decoloratus* has been experimentally carried from egg to egg in twenty-five days but it is much influenced by climate and season. The larvæ are known as "seed ticks."

Relation to Disease.—*Boophilus* is the carrier of *Piroplasma bovis* (=*bigeminum*), the cause of bovine piroplasmosis, known under various names as Texas fever, tick fever, splenetic fever, etc. Fever, emaciation, anæmia, bloody urine and enlarged spleen are the usual symptoms, and they commence about ten days after infestation. The mortality may be very high (40 to 100 per cent in the case of *B. decoloratus*). "Salted" animals and those cured by treatment continue to harbour the Piroplasma and act as "reservoirs."

Treatment.—The intravenous injection of 200 c.c. of 1·5 per cent aqueous solution of trypanblue has proved very successful.

Eradication of Ticks.—The double method of cleaning the animals and starving out the ticks in meadows by "pasture rotation" has been particularly successful in the case of this tick, largely on account of its one-host habit of life. Extensive tracts have been cleared of the tick by these means in the United States.

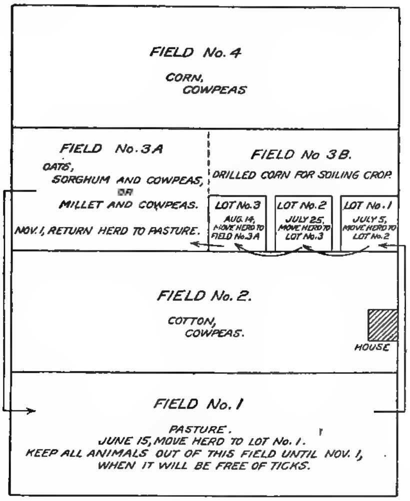

FIELD No. 4
CORN, COWPEAS
FIELD No. 3A
OATS, SORGHUM AND COWPEAS, OR MILLET AND COWPEAS.
NOV. 1, RETURN HERD TO PASTURE.
FIELD No 3B.
DRILLED CORN FOR SOILING CROP.
LOT No. 3
AUG. 14, MOVE HERD TO FIELD No. 3A
LOT No. 2
JULY 25, MOVE HERD TO LOT No. 3
LOT No. 1
JULY 5, MOVE HERD TO LOT No. 2
FIELD No. 2.
COTTON, COWPEAS.
HOUSE
FIELD No. 1
PASTURE.
JUNE 15, MOVE HERD TO LOT No. 1.
KEEP ALL ANIMALS OUT OF THIS FIELD UNTIL NOV. 1, WHEN IT WILL BE FREE OF TICKS.

Fig. 139.—Scheme for clearing a farm from *D. venustus* in 4½ months. (After H. W. Graybill.)

Tick-infested cattle may be cleared of the parasites by dipping or spraying. Formerly kerosene emulsions were largely used for this purpose, but arsenic preparations have been found more effective and less injurious to the skin (see p. 489).

Pasture rotation depends on the following principle. The shortest time in which "seed ticks" will appear after engorged females have fallen to the ground is twenty days, so that for that length of time cattle are safe from re-infestation from their own ticks. Secondly, all the ticks will drop off, gorged, from an infected beast within a period which varies, according to the time of year, from five to ten weeks. Infested cattle, therefore, are placed on tick-free pasture and left there for twenty days and then moved on to a second, and if necessary to a third field, not more than twenty days being allowed in each. All the ticks will now have fallen off, and if it is so arranged that no animals are allowed in these fields for a known length of time, depending on climate and season, but carefully determined for each district, all the ticks on them will die of starvation. Various schemes have been worked out combining pasture rotation with rotation of crops, and by choosing suitable dates it is found possible to clear a farm from ticks in less than six months. A scheme devised by the Bureau of Animal Industry of the United States to clear a farm in four and a half months is here inserted in illustration of the method employed, though it concerns another tick—*Dermacentor venustus*.

Longirostrata

Genus 8, **Hyalomma.**—It is unnecessary to characterize this genus, as for economic purposes it may be said to contain a single species, *Hyalomma ægyptium*. It is noteworthy for its dark-brown scutum with hemispherical eyes, which are not marginal as in most ticks but internal to the scutal border, and its banded legs. The male has a special ventral armature, there being, in addition to anal plates somewhat like those of *Rhipicephalus*, a pair of chitinized points borne on posterior abdominal protrusions.

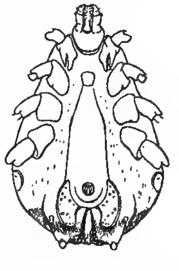


Fig. 140.—*H. ægyptium*, ♂, showing anal armatuer. Much enlarged. (Nuttall and Warburton.)

This is one of the commonest ticks on domesticated and many wild animals throughout Africa and India. It also occurs in South Europe and various parts of Asia.

Life-history.—This is sometimes a two-host, sometimes a three-host tick. Very many eggs are laid (up to 15,000), which take thirty-five days to hatch at 18° C. The larvæ gorge in from four to fifteen days, when some drop off, others remaining on the host to change to nymphs. Females remain on the host for about a week and are fertilized there, the males remaining anchored often for months. The life-cycle may be completed in 116 days, but fasting larvæ and adults are very tenacious of life. Nymphs live no more than ninety days without food, but larvæ have been kept alive for 369 days and females for 817 days (Nuttall). It is known at the Cape as the "bont legged tick."

Relation to Disease.—There is at present no definite proof that *H. ægyptium* is pathogenic, but there is a strong suspicion that it transmits *Nuttallia equi*, the cause of biliary fever in horses, there being a remarkable agreement between the distribution of the species and the incidence of the disease in Africa, Asia and South Europe. In any case the tick is so abundant as to be distinctly injurious to domesticated animals

GENUS 9, **Amblyomma.**—*Generally ornate, with eyes and festoons. Palps long. Males without adanal shields.*

Many species of *Amblyomma* have been described, and from a great variety of hosts. A few affect only cold-blooded animals, but in general the host relationship is remarkably indefinite, almost any animal being attacked. The coloration, especially of the males, is often quite beautiful, green, red and metallic tints occurring in lines and patches on the dark background of the scutum. There are no European species, but America, Africa and Asia are well represented and there are some Australian forms. Only a few occur so abundantly as to possess economic importance, and only one, *A. hebræum*, is so far known to be pathogenic. The life-history is known in very few cases, and these all prove to be three-host ticks.

A. cajennense (the "Cayenne tick") is wonderfully abundant in Central America and overflows into Texas, where it is a great annoyance to domestic animals and to man. It seems impossible to control a tick which practically attacks anything, and the only resource is dipping and spraying. The same may be said of *A. americanum*, the "Lone Star" tick, so called on account of the silver spot at the apex of the female scutum. It occurs throughout the eastern United States—especially in those States bordering on the Atlantic and the Gulf of Mexico. Young chickens have been killed in massive attacks by its larvæ.

A. variegatum is very common on cattle and other animals in Africa, but does not appear to do more than cause annoyance.

A. hebræum, the "bont-tick," is a South African species.

Life-history.—A large number of eggs (up to 18,500) are laid on the ground, and hatch in about fifty days. The larvæ feed for five or more days and drop off. The nymphs feed for about a week, and in their turn drop off. Pairing takes place on the host, where the males await the females. The life-cycle has been completed in 171 days. Fasting larvæ may live for 346 days, nymphs for 250 days, and adults for two years. Gorged females attain a very large size—up to 3 cm. in length.

Relation to Disease.—*A. hebræum* transmits to sheep, goats and cattle in Africa a disease known as "heart-water," due to an undetermined and probably ultramicroscopic organism.

Eradication.—Frequent dipping or spraying is practically the only measure. It has been very successfully practised in parts of South Africa.

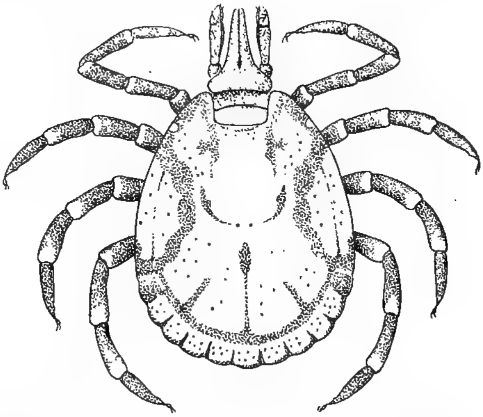


FIG. 141.—*A. hebræum*, ♂. Greatly enlarged, showing the dorsal pattern. (Original.)

GENUS 10, **Aponomma.**—Differs from *Amblyomma* only in being eyeless and generally rather broad in proportion to its length, the scutum being often broader than long. These ticks are, as a rule, small in size and variegated with metallic markings. Though occasionally found on mammals they are normally parasitic on reptiles, and are therefore of no economic importance.

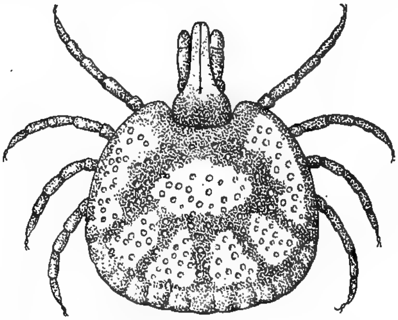


FIG. 142.—*Aponomma gervaisi*, ♂. Much enlarged. (Original.)

In several species of *Amblyomma* the eyes are degenerate and not very easy to see, and there are all gradations of form between the two groups, so that, although there is a certain convenience in separating the eyed forms which attack mammals from the blind forms parasitic on reptiles, the generic distinction is hard to maintain, and it would perhaps be better to consider *Aponomma* as a sub-genus of *Amblyomma*.

List of Ticks known to be Pathogenic

Tick.	*Distribution.*	*Disease conveyed.*
Argas persicus	Cosmopolitan in warm countries	Spirochætosis in fowls (*S. gallinarum*).
Ornithodorus moubata	Africa	Relapsing Fever in man (*S. duttoni*).
Ixodes ricinus	Europe, N. America, etc.	Bovine Piroplasmosis (*P. divergens*).
Hæmaphysalis leachi	Africa, Asia, Australia	Canine Piroplasmosis.
H. cinnabarina, var. *punctata*	Chiefly Europe	Bovine Piroplasmosis.
Dermacentor venustus	Rocky Mountains	Spotted fever in man, and "tick paralysis."
Rhipicephalus bursa	Mediterranean region	Ovine Piroplasmosis ("Carçeag").
R. appendiculatus	Africa (Central and Southern)	Rhodesian or E. Coast Fever in cattle (*Theileria parva*).
R. sanguineus	Cosmopolitan	Canine Piroplasmosis
R. simus	Africa	Rhodesian or E. Coast Fever in cattle.
R. evertsi	Transvaal	Equine Piroplasmosis.
Boophilus spp.	Cosmopolitan	Bovine Piroplasmosis (*P. bovis*) (=*bigeminum*).
Amblyomma hebræum	S. Africa	Heartwater in cattle.

Species suspected to be Pathogenic

Tick.	*Distribution.*	*Disease conveyed.*
Argas reflexus	Europe and N. Africa	Spirochætosis in fowls.
Ixodes pilosus	Cape Colony	Paralysis in sheep.
Rhipicephalus capensis	S. Africa	Rhodesian or E. Coast Fever in cattle.
Hyalomma ægyptium	Africa, S. Europe, India.	Equine Piroplasmosis.

The first two have been experimentally proved capable of transmitting the diseases in question.

Species of some Economic Importance though not Pathogenic

Tick.	*Distribution.*	*Host.*
Ornithodorus megnini	Mexico and S.W. United States	Ears of horses and mules.
Hæmaphysalis bispinosa	Southern Asia.	All domestic animals.
H. inermis	Transcaucasia	Cattle.
Dermacentor reticulatus	Europe and W. Asia	All domestic animals.
D. nitens	Tropical America	Ears of horses and mules.
D. variabilis	United States	Dogs.
Amblyomma cajennense	Central America	Domestic animals and man.
A. variegatum	Africa	Cattle.

Tick Paralysis

A disease known as Tick Paralysis occurs extensively in sheep in Cape Colony. Animals lag behind the rest of the flock, lie down, and are soon unable to rise. Out of doors they are liable to become the prey of jackals, but under shelter, spontaneous recovery follows rapidly in most cases. There is no fever, and no noticeable lesions are found on autopsy.

The disease is locally attributed to *Ixodes pilosus*, which, however, is found on both healthy and affected animals. Inoculation experiments gave no results. Dipping the sheep had a marked beneficial effect, both as preventive and cure (Borthwick).

Hadwen has noticed a similar sheep disease in British Columbia, and was able to produce it experimentally by means of the tick *Dermacentor venustus*. Hadwen and Nuttall also produced the disease in a dog, through the same tick.

Todd and Temple have recorded cases of alleged tick paralysis in man in British Columbia and Oregon. Here there were febrile symptoms, temperature 104°, protruding tongue, mottled skin, etc. In most cases recovery followed rapidly after removal of ticks (apparently *D. venustus*) from the head or neck. Nuttall remarks that it is extremely common to have that region infested by ticks, and that too much importance should not be attached to finding a tick or two on the head, since normal persons would probably be found to be similarly infested.

Nevertheless, the experiments of Hadwen and Nuttall prove that tick paralysis exists as a definite affection, which, however, appears to be communicable by entirely different species of tick (*D. venustus* and *I. pilosus*).

Dipping as a Protection against Ticks

Experiments on a considerable scale have been made by the Bureau of Animal Industry in the United States and by Watkins-Pitchford and Cooper and Laws in South Africa in the use of arsenical dips for tick-infested cattle. It is established that the effect on the tick is toxic and not merely repellent, and that in addition to the high mortality inflicted on the ticks by dipping, the oviposition of those which survive is to some extent interfered with and a proportion of their eggs fail to hatch. Dipping protects the animals from reinfestation for a day or two unless exposed to rain.

About 1 lb. sodium arsenite is added to 50 gals. water, giving 0·16 per cent of arsenious acid. Cooper and Laws found that the addition of an emulsion (soap, oil, glue) greatly increased the effectiveness of the dip, 0·153 per cent As_2O_3 with emulsion being equivalent to 0·225 per cent As_2O_3 without. In the experiments of Cooper and Laws 400-600 cattle were dipped per hour at an expense of $\frac{1}{8}$-$\frac{1}{4}$d. per head. *Boophilus* was easily killed, but *A. hebræum* was very difficult to destroy, and the dip had to be strong and applied at intervals of not more than a fortnight. Against *R. appendiculatus* three-day intervals were necessary (see Cooper and Laws on the Theory and Practice of Dipping, *Parasitology*, vol. viii. p. 190).

Determination of Genera in the Ixodidae

The gorged larvæ, nymphs and females of all Ixodid ticks look much alike, though of course they are much larger for a large species like *Amblyomma hebræum* than for a small species like *Hæmaphysalis bispinosa*. Slight differences of coloration when seen in the mass are noticed by those who are familiar with them as parasites of domestic animals, hence such local names as the "blue tick" (*Boophilus*), the "red tick" (*R. evertsi*), the "brown dog-tick"

(*R. sanguineus*), the "bont tick" (*A. hebræum*), etc. These, however, are of little use in determining the tick, and the parts which must be carefully examined form but a small portion of the whole distended mass. It is better to select only slightly swollen examples for examination.

If the tick is six-legged it is a larva. If it is eight-legged but has no sexual aperture (which is usually between coxæ II.) it is a nymph. A nymph is also distinguished from a mature female by the absence of "porose areas" (see Fig. 128, B).

The determination of the species is of course beyond the scope of the present article, and the aid of an expert will often be necessary, but if the genus be recognized, and the host and the locality taken into consideration, the field is at all events much narrowed down, and in many cases there will be little doubt as to the actual tick in question. If we omit *Rhipicentor* and *Margaropus*, which are exceedingly unlikely to be met with, it will not be difficult to discover to which of the other eight genera our specimen belongs.

Suppose the specimen to be a male, in which case the whole of the back will practically be covered by the scutum. If the tick be *ornate* (with white or coloured markings on the scutum) the genus is either *Dermacentor*, *Amblyomma* or *Aponomma*—or just possibly *Rhipicephalus*, of which there happen to be a very few ornate species. If *Rhipicephalus*, it will have anal plates; if *Dermacentor*, coxa IV. will be enlarged; *Aponomma* is eyeless.

Thus we have:

Ornate Males

1.	No eyes	*Aponomma.*
	Eyes	2.
2.	Anal plates	*Rhipicephalus.*
	No anal plates	3.
3.	Coxa IV. enlarged	*Dermacentor.*
	Coxa IV. normal	*Amblyomma.*

If *inornate*, the specimen may belong to any genus, for there are inornate examples of *Dermacentor*, *Amblyomma* and *Aponomma*, while *Rhipicephalus* is generally without scutal pattern. The eyes, again, may help us at first. *Ixodes*, *Hæmaphysalis* and *Aponomma* are eyeless; the rest are eyed. The eyeless genera are easily distinguished; *Ixodes* has no festoons; *Hæmaphysalis* has characteristic palps. The five remaining eyed genera are less easy, but the anal plates again aid us. Luckily, we may eliminate *Dermacentor*, for the only inornate example of that genus likely to be met with is sufficiently indicated by its habitat. It is *D. nitens*, which infests the ears of horses and mules in tropical America. There remain, then, *Rhipicephalus*, *Boophilus*, *Hyalomma* and *Amblyomma*. The last-named has no anal plates. *Hyalomma* has long palps and its hemispherical eyes are within the margin of the scutum. *Boophilus* has four anal plates of fairly equal size, while the outer plates are either small or absent in *Rhipicephalus*. Thus:

Inornate Males

1.	Eyeless	2.
	Eyed	4.
2.	No festoons	*Ixodes.*
	Festoons	3.
3.	Palps conical (never ornate)	*Hæmaphysalis.*
	Palps long and rod-like (generally ornate)	*Aponomma.*
4.	Palps long	5.
	Palps short	6.
5.	Anal plates	*Hyalomma.*
	No anal plates	*Amblyomma.*
6.	Four anal plates fairly equal in size	*Boophilus.*
	Outer anal plates small or absent	*Rhipicephalus.*

As anal plates are never present in the female, and the festoons vanish on gorging, these indications of the genus are no longer at our disposal in the case of a female tick. The following table may be of assistance:—

Females

1.	Eyeless	2.
	Eyed	4.
2.	Anal groove in front of anus ⌒ (prostriate)	*Ixodes.*
	Anal groove behind anus ⌣ (metastriate)	3.
3.	Palps conical, broadest at art. 2 (never ornate)	*Hæmaphysalis.*
	Palps long and rod-like (generally ornate)	*Aponomma.*
4.	Basis capituli hexagonal, pointed at the sides	5.
	Basis capituli otherwise	6.
5.	Palps moderately short; spiracle, comma-shaped	*Rhipicephalus.*
	Palps very short and ridged; spiracle circular	*Boophalus.*
6.	Palps rather short and massive (generally ornate)	*Dermacentor.*
	Palps long	7.
7.	Inornate, eyes not marginal	*Hyalomma.*
	Generally ornate, eyes marginal	*Amblyomma.*

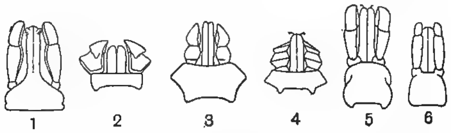


Fig. 143.—Diagrams of typical capitula in the principal genera.

1, *Ixodes*; 2, *Hæmaphysalis*; 3, *Rhipicephalus*; 4, *Boophilus*; 5, *Hyalomma*; 6, *Amblyomma* and *Aponomma*.

C. W.

PARASITIC INSECTA

Insects are air-breathing arthropoda. They are differentiated into head, thorax, and abdomen, the head carrying a pair of antennæ, and the thorax carrying three pairs of legs and often two pairs of wings. Some, however, are wingless.

Two orders furnish parasitic members, viz. **Hemiptera** or **Rhyncota, Diptera.**

Hemiptera

These are suctorial insects, usually with two pairs of wings, the anterior pair variable, often half leathery and half membranous, the posterior pair membranous. Metamorphosis is very incomplete.

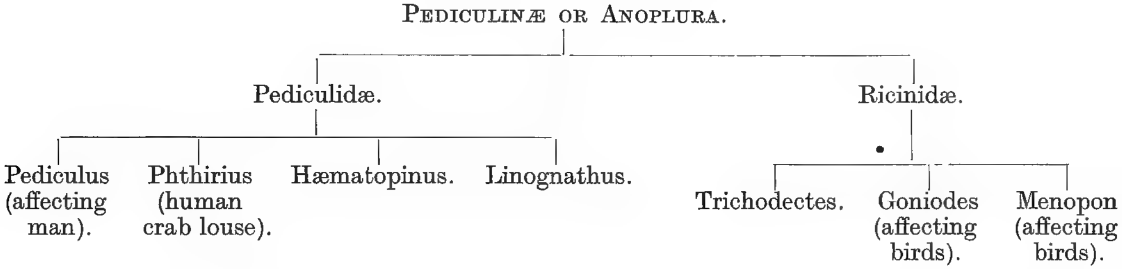
The *Hemiptera* include the *Pediculinæ*, which furnish the various lice or pediculi.

Pediculi or *Lice* have a projecting rostrum with barbed hooklets and with a hollow extensile sucker. They have simple eyes, no wings, undergo no metamorphosis. The antennæ are five-jointed; the feet have hook-like end joints, and the barrel-shaped eggs (nits) are deposited on the hair of the host.

In the **Pediculidæ** (*Hæmatopinus* and *Linognathus*) the thorax is larger than the head but smaller than the abdomen. The mouth is at the extreme anterior end, is intended for puncturing and sucking, and is armed with spines by which to fix itself to the skin. Tarsi end in a claw. *Hæmatopinus* and *Linognathus* affect all the domesticated animals except the cat.

In the **Ricinidæ** (*Trichodectes*) the head is massive and square—larger than the thorax. The mouth is on the under surface of the head and is intended for mastication (the parasites being pellivorous). They are not blood-suckers (so more like commensals than parasites) but cause irritation and pruritus. The tarsi end in two claws. *Trichodectes* affect all the domesticated animals except the pig.

Goniodes and *Menopons*, with some other members of the family, affect only birds.


PEDICULINÆ OR ANOPLURA.
Pediculidæ.
Ricinidæ.
Pediculus (affecting man).
Phthirius (human crab louse).
Hæmatopinus.
Linognathus.
Trichodectes.
Goniodes (affecting birds).
Menopon (affecting birds).

Lousiness; Phthiriasis; Pediculosis

This includes all infestations by the apterous insects which do not jump like fleas; and except the *Melophagus* of the sheep they all belong to the *Pediculinæ*. The representative sub-genera of these are the *Hæmatopinus* and *Linognathus* and *Trichodectes*. The various species are specific for the animal (host) they affect, and will not live and propagate on other species of animals.

Horse : (3) *Hæmatopinus macrocephalus* (*H. asini*), the commonest; *Trichodectes pilosus*; and *Trichodectes pubescens* (*T. parumpilosus*) more rare.

Ox : (3) *Hæmatopinus eurysternus*; *Linognathus vituli* (*H. vituli* or *H. tenuirostris*); *Trichodectes scalaris*.

Sheep : (2) *Linognathus pedalis*; *Trichodectes sphærocephalus* (*T. ovis*), and this rare. Much commoner is the Dipterous insect *Melophagus ovinus*—which is a degenerated and apterous insect, living on fat and blood by bites; like a louse.

Goat : (2) *Linognathus stenopsis*; *Trichodectes climax*.

Pig : (1) *Hæmatopinus urius* (*H. suis*), the largest of *Pediculinæ*.

Dog : (2) *Hæmatopinus piliferus* (*Linognathus piliferus*) and *Trichodectes latus* (the host of the cystic stage of the tapeworm—*Dipylidium caninum*).

Cat : (1) *Trichodectes subrostratus* (very small).

Symptoms.—Differ with the genera in that the *Hæmatopinus* punctures the skin and sucks blood, whereas the *Trichodectes* does not, but even this parasite causes a good deal of irritation.

With both there is great pruritus and consequent rubbing and scratching. The skin is irritated and the epidermis desquamates a good deal, *i.e.* much scurf or dandruff, and the hair looks coarse and erect. Small rough crusts of dried blood and scurf may often be found. If neglected, lice reduce an animal's condition greatly and cause emaciation, anæmia and debility, and may prove fatal. Dirt, neglect and malnutrition predispose to infestation with lice and prevent their removal.

Lice are more active in warm weather, or on animals housed; they do not show any tendency to leave the host (apterous). On careful examination at the root of the hair one can find parasites and eggs (nits).

In the horse the parasites are usually located about the mane and root of the tail, but if neglected the parasites spread further. The hair is rough, erect, matted, rubbed off in patches. There is much scurf, and the parasites, nits, dead bodies, etc., are easily found. Pruritus is marked with *Hæmatopinus*, less so with *Trichodectes*.

In cattle *Trichodectes* (more common) are spread over the whole trunk, whilst *Hæmatopinus* chiefly affects the head, ears and neck and upper surface of back and loins (in fact, along the spine).

In sheep the *Melophagus* is found at the bottom of the fleece—at surface in hot weather—indicated by pupæ (like apple pips) and parasites. In shorn sheep the parasite is found where the sheep cannot reach it—about the ears and in front of the shoulders. It causes great pruritus and the sheep bite and gnaw themselves. *Trichodectes* are rare, but do much harm by gnawing through the wool and so causing bare patches.

Pigs are seriously affected by presence of lice. The skin is often excoriated, pruritus is intense and the animal rubs against anything. Young pigs neglected may die.

In dogs lice are often responsible for very serious effects. When in small numbers they are mainly found at the back of the head and about the ears, and also at the base of the tail. In heavy infestations lice and their eggs or nits are present in enormous numbers all over the body surface. They cause great irritation and constant scratching, rubbing, and rolling on the ground, and in some cases intense anæmia which may have fatal results. The blood-sucking louse (*Hæmatopinus*) is easily seen, bluish in colour from ingested blood and with its head buried into the skin. The *Trichodectes* may not be observed so readily amongst the scales of dandruff without the use of a hand lens or microscopic examination.

In cats the very small *Trichodectes* are not easily seen amongst the scurf to which they give rise, and magnification is often necessary for their detection. Cats lose condition through lack of rest when the lice are numerous, but the effects are rarely so severe as those produced in dogs by the blood-sucking lice.

Dogs and cats may become infected with tapeworm (*Dipylidium*) as the result of nibbling lice, as the *Trichodectes* frequently harbour the intermediate stage of that parasite.

Lice of several species affect fowls and pigeons—in fact all kinds of birds.

Treatment in Animals consists in :

(1) Cleanliness: grooming and cleaning, clipping ;

(2) Good feeding, which is necessary to combat effects of a widespread infection ; and

(3) Application of some parasiticide. Whatever dressing is used must be persisted in and repeated thoroughly for several weeks in order to destroy the fresh lice as they are hatched out. The eggs or nits are extremely resistant to any dressings that can be used with safety. Vinegar is often added to penetrate nits.

The various agents used are : Stavesacre seeds (a decoction of bruised seeds 1-20, with some soft soap added to it).

Tobacco (crude gardener's tobacco) 1 oz. to 1 oz. water—making a decoction (water just simmering).

Creolin 3-5 per cent.

Paraffin oil 1 in 20 in linseed oil.

Sheep dips. Mercurial ointment. Chinosol (1-500). Eusol. Sol. picis carbonis.

An effective dressing for lice can be made from :

Sulphur 1 lb.
Hellebore 1 lb.
Tobacco ½ lb.
Cofectant ℥ii.

Allow the tobacco to simmer in some water till all the juice is removed, then strain and press. Then add the sulphur, hellebore and cofectant, stir well, and fill up to 2 gallons with water. Apply well with a brush and wash off in four days.

Various insecticide powders may be used : pyrethrum, lobelia, stavesacre, cevadilla ; hellebore, sulphur.

Affected animals should be isolated, and stalls, boxes, kennels, harness, rugs and utensils should be disinfected as in mange.

Diptera

Diptera are suctorial insects possessing only two wings which are membranous, the posterior pair of wings being replaced by two balancers—slender stalks with a knob at the end. They have a proboscis or sucker which is often used for puncturing, and they possess salivary glands which in many cases secrete a poisonous irritant liquid.

In the course of development diptera undergo a " complete metamorphosis " consisting of three stages besides the egg. The adult female or perfect insect (the imago) after fertilization deposits its eggs, which under favourable conditions hatch and liberate apodal larvæ (maggots). Each maggot develops into a pupa (nymph or chrysalis), which in turn develops into the imago or adult forms.

The parasitic diptera are included in three sub-orders, namely :

1. *Aphaniptera* (fleas).
2. *Nemocera* (mosquitoes, gnats, sandflies).
3. *Brachycera* (the so-called " true flies ").

Fleas (*Pulicidæ*)

Fleas are jumping insects, with three pairs of long powerful legs, body flattened laterally ; wingless ; three segments of thorax and distinct abdomen with nine segments. The buccal apparatus of the adults is adapted for piercing and suction. Length, males about $\frac{1}{16}$th inch, females about $\frac{1}{12}$th inch ; colour brown. Fleas are parasitic on mammals and birds and live by blood-sucking. The adult female deposits about 20 white barrel-shaped eggs, generally in dusty corners, cracks in floors and walls, etc. In from six to ten days the white larvæ or maggots with 14 segments hatch out. In about ten days the larva spins a cocoon, moults and becomes a hexapod or six-legged pupa, and this in from ten to twenty days more develops into the adult stage. In some instances the whole development may occur on the body of the host—often seen on cats.

Fleas are mainly parasitic on man, dog, cat, rabbit and bird, the species affecting man being *Pulex irritans* ; and dogs and cats, *Pulex serraticeps*.

Symptoms.—Seen mainly on young animals kept several together, more or less in confinement. Fleas cause constant irritation with biting and scratching. When very numerous they cause general debility, both by blood-sucking and preventing rest. If the whole evolution occurs on the host, as is not infrequent in cats, there is often an eruption and skin irritation lasting for weeks, giving rise to mistaken diagnosis of eczema or mange.

The dog flea may be the intermediate host of the tapeworm *Dipylidium caninum*, and also of certain small nematode worms found in the blood of the dog.

Treatment.—Preventive measures consist of general cleanliness, washing with antiseptic soap, frequent changing or disinfection of bedding. Affected animals may be dressed with an infusion of stavesacre (1 in 20 of boiling water, allowed to stand for 24 hours and then strained); sol. picis carbonis (1 or 2 per cent), eusol, creolin; or dusted with such insecticide powders as pyrethrum, lobelia, and hellebore. The latter is very poisonous and should be used with care and well diluted. Infected beds and bedding should be destroyed or sterilized and kennels and floors thoroughly disinfected.

Diptera Parasitic in Larval State on the Skin

Of the sub-order *Muscidæ*, the two genera *Sarcophaga* and *Lucilia* furnish members which deposit their eggs on the skin of animals and so provide their larvæ, which are obligatory parasites, with a suitable host.

In many parts of Europe *Sarcophaga magnifica*—the *magnificent sarcophagus*—deposits its eggs in wounds of man and animals. A small wound only is needed, or a moist surface; *e.g.* a mucous membrane, or perineum, or in inguinal region, or prepuce, or sometimes in dog's ears. The larvæ soon increase the size of the wound, or ulcerate a sound surface and may do very serious damage. Cattle are most commonly affected.

Treatment.—Pick off and destroy the parasites and disinfect the wounds.

Maggot in Sheep.—In the British Isles various species of *Lucilia* are the chief agents acting in this way—*Lucilia cæsar*, or more commonly *Lucilia sericata*—and in both North and South America *Lucilia macellaria*. These deposit eggs in wounds of man and animals, or sometimes in the natural orifices—nostrils, anus, vulva and prepuce and soiled perineum. In sheep, especially, the condition known as "The Fly," "Fly-blown," "The Maggot," is common.

The flies are much like the common blue-bottle; are green or greenish-blue in colour, have soft probosces and cannot puncture the skin, but deposit eggs on the surface. A certain amount of moisture, dampness of the surface, is necessary to their development; so the condition is found in damp, low-lying pastures, in muggy weather, and on sheep where the fleece is wet with fæces or urine. Hence it occurs in sheep with dirty long tails and hind-quarters, or with a wound somewhere, as after shearing. The larvæ are produced very rapidly (may be even on the same day); are white to cream-coloured maggots, with two hooks on the tapered head.

Symptoms of Maggot in Sheep.—From the irritation set up the sheep are seen to be constantly shaking or wriggling the tail. Often when feeding they suddenly dart forward, and wriggle the tail, and may try to bite the part or to rub against posts or gates. On inspection one finds wool dirty and matted, and under it a swarming heap of maggots which have penetrated skin and tissues, often undermining skin a foot or more, and sometimes penetrating even into the peritoneum. There is great wasting, and the condition is often fatal if untreated.

Treatment: (*a*) *Preventive*—avoid the damp and dirty predisposing conditions by *docking* and *clipping long wool of hind parts* (locking), or by applying a dressing to keep away the fly—*powdering with sulphur*, or *dipping the sheep*. Sheep or lambs with wounds after shearing should be carefully watched and, if necessary, brought up at night.

(*b*) *Curative.*—The affected sheep should be brought up and have all the maggots brushed or picked off and destroyed by fire or by a strong disinfectant solution in hot water, then the part washed with an antiseptic solution—creolin, corrosive sublimate, or copper sulphate. Turpentine and paraffin are also useful in weak dilution in oil (1-10). The diet must be generous.

Ox Warble Flies, Horse Bot-flies, and Sheep Nostril Flies are all included among the Diptera in the same family, Œstridæ. In this family the adult flies have the following common characteristics: They are thick-set and hairy, the head is large and the eyes are well separated. The mouth parts are rudimentary and the insects seldom feed and neither bite nor sting. The antennæ are short and three-jointed. The thorax presents a distinct transverse groove. The abdomen is short and ends in the female in a long ovipositor. The wings resemble those of the house-fly.

Oestridæ are exclusively parasitic in habit. The female lays her eggs on the body of the special host to which that species is attached, and the larvæ by various means reach their special habitat; *e.g.* subcutaneous tissue in case of *Hypoderma bovis*, stomach in case of *Gastrophilus equi*. Some species are viviparous, producing larvæ directly. The larval state is the parasitic one; and after a certain time the larva quits its host, becomes a pupa or nymph, and after a period of four to eight weeks the perfect insect is produced. The two genera of importance are *Hypoderma* and *Gastrophilus*.

Hypoderma.—There are two important species of the genus hypoderma: *Hypoderma bovis* or *Œstrus bovis*, the common one in Britain and in Europe, and *Hypoderma lineata* (striped hypoderma), less common in Europe; also found in America.

Ox Warble Fly; "Gadfly" (*Hypoderma bovis*; *Œstrus bovis*).—The perfect insect, a little bigger than ordinary housefly—up to 15 mm. or $\frac{3}{5}$ths inch long—has a black body with various coloured hairs on abdomen, yellow in front, black in middle, and orange-coloured at posterior end. The female has a long ovipositor. Wings are brownish.

The insect is found in July, August to early September; makes a buzzing noise whilst flying; darts down on to the limbs, shoulders or neck of an ox and deposits an egg on the skin, then up and down again, and so on for 10 or 12 eggs. The cattle are usually in a state of terror when this fly is about—galloping wildly with tails erect and bellowing—called "gadding"—and are only safe and quiet if they can wade out into a pool or stream. A curious fact is that the oviscapt of the fly is soft and cannot pierce the skin, and therefore there is little chance of any pain being caused. The eggs are white and elongated and have at one end a brownish substance which fixes them to the hair of the host. The *larvæ* or *warbles* are the cause of serious symptoms, for they are found under the skin of the host in the subcutaneous tissue. The older view with regard to their method of reaching this situation was that the newly hatched larvæ, produced from eggs laid on the skin of the back, burrowed through under the skin, and that the small puncture so made was unnoticed or obliterated. The view has also been held that either the eggs deposited on the hair of the limbs and sides of cattle, or freshly hatched larvæ on the skin, are licked off by the host and find their way into the alimentary tract and then, by migration, in a minute form reach the subcutaneous tissue. Migration forms have been found.

Carpenter has conclusively shown that the larvæ actually burrow through the thin skin of the upper part of the limbs and thence migrate to their predilection seats under the skin of the back.

During the winter no symptoms develop, but in the spring nodular swellings are seen along the *back*; a variable number, even as many as a hundred, and about the size of half a walnut. They are not very painful nor hot. There is a small hole at the summit, close to which is applied the anal end of the larvæ with its two stigmata—breathing pores. The swelling contains simply the larva, pus, and often blood, and the tissues round are somewhat inflamed. As the larva grows a little liquid is often exuded from the opening and mats the hair, but the hole gradually enlarges till it is sufficient to allow the exit of the larva—mature—in June or July. The hole then closes up and the swelling gradually subsides; but the hole in the skin remains as a blemish and hole whenever the host (ox) is killed and its hide tanned. The escaped larvæ bury themselves in the soil or under hay or leaves, etc., become nymphæ, and in about one month perfect insects.

Larvæ nearly mature can easily be squeezed or pressed out of the swellings and are brownish or brownish-black in colour, with body made up of eleven rings or segments, pyriform or conical in shape, with numerous spines arranged in three rows on each side. The mouth has on each side a sharp hook directed almost at right angles to the body, and these hooks fix the head of the parasite and also help the mouth organs in boring into the tissues.

If many warbles are present there is irritation, malnutrition, emaciation; but much worse is the serious loss through damage to hides, and loss of milk and flesh during the "gadding" season.

Treatment.—During the early stages one may apply any antiseptic astringent lotion; later one may apply (1) an oily dressing over the hole to asphyxiate parasite, (2) ointment containing tar or creosote, or (3) ung. hydrarg. or (4) squeeze out parasite even after an incision, which helps the healing process. Care must be taken not to burst the maggot in attempting to squeeze it out, as if this is done an abscess is likely to be produced.

Preventive Measures.—(1) Destroy all larvæ which come out, for flies are only got from these. (2) Apply a dressing to cattle in July and August to prevent fly attacking them: Stockholm tar, sulphur and lard—two or three applications; or sulphur 4, ol. picis 5, and oil up to 27 oz.; or strong NaCl and alum solution applied oftener (weekly)—all along backs of cattle.

Hypoderma equi or "**Hypoderma Loisetti.**"—Fairly common in Holland and Belgium and in north of France; rarely in Britain. The tumours are very similar to those of the ox, but the larvæ are much smaller, only ½ inch long, with no spines and no oral hooks. The skin is thin and semi-transparent. The lesions are met with chiefly on withers and back and so interfere with harness.

It has been held by Joly that the larvæ found in warbles met with in the horse belong to a separate species of *Hypoderma*, although the adult fly has not been reared out from the maggots. Railliet in France, and Megnin identified some specimens as the larvæ of the *Hypoderma bovis* in the penultimate stage. J. F. Craig was able to secure two batches of larvæ, one taken from a military horse and the other from a four-year-old dun pony treated in the Dublin College clinique. The latter animal had about a dozen warbles scattered over the sides of the abdomen and thorax and the back and loins. They were about the size of hazel nuts, fairly well defined and not very painful. The hair over them had become agglutinated with a glairy yellowish-white pus, which had evidently escaped through a small opening at the summit of each of the swellings. After enlarging the openings a small maggot, together with some pus, was squeezed out of each of the warbles. These larvæ were whitish in colour, somewhat spindle-shaped, with blunt extremities; they varied in length from 12 to 15 mm. and were provided with a thin, almost transparent, cuticle. They differed from the larvæ of the horse warble described in Neumann's *Parasites*, amongst other things by the possession of spines on the rings. Prof. Carpenter, Dublin, examined these specimens and identified them as the third-stage larvæ of one of the *Hypoderma* of the ox, whether *Hypoderma bovis* or *lineata* he was unable to state. It is quite possible that if the larvæ in the warbles of the horse were more generally examined, the ox *Hypoderma* might be found to be the only, or at least the chief, fly responsible for the production of warbles in equines. That the larvæ were only third-stage larvæ and had not attained maturity might account for the failures previously experienced in raising the adult fly from the maggots.

The treatment for warbles in the horse is similar to that for cattle.

G. H. W.

Horse Bot-flies belong to the genus *Gastrophilus* in the family of Œstridæ. They are large hairy flies in which the mouth parts are rudimentary. The antennæ are short, three-jointed, carrying a three-jointed arista. Between the large compound eyes are three ocelli. The thorax has a distinct transverse groove. The female has a long ovipositor, bent under the body when the insect is at rest. The larvæ of this genus are parasitic in the stomach, pharynx, œsophagus, duodenum, and rectum of equines.

The genus contains fourteen species, of which the more common are: *Gastrophilus intestinalis*, De Geer; *G. nasalis*, Linné; *G. hæmorrhoidalis*, Linné; and *G. pecorum*, Fabr.

Gastrophilus intestinalis, De Geer (1776) (*Gastrophilus equi*, Clark, 1797), is the most common bot-fly of equines and is widely distributed throughout the world.

The adult fly is about 12 to 15 mm. long. The body is very hairy, the head light brown with a white pile in front, thorax black covered with reddish hair with a transverse band of black hairs behind the suture. Abdomen is reddish brown with rows of black spots at the posterior borders of the segments. The wings are transparent with a dark transverse band and two dark spots at the apices.

The males are very rare and seldom seen.

The adult flies appear during the summer and autumn. They live only a short time, they partake of no food and are chiefly engaged in procreation. The females after coupling are often seen in the warm sunshine hovering near equines. When an egg is ready to be laid, the fly darts towards the animal and fixes the egg to the hair and then flies off until another egg is ready, when the process is repeated. A female fly lays as many as 400 or 500 eggs. The presence of the fly and the process of egg-laying in no way disturb the horses. The eggs are deposited commonly on the hairs of the fore limbs, the shoulders and chest, where they can easily be reached by the animal's tongue. They appear as yellow specks firmly fixed to the outer portions of the hairs and often arranged closely together. Each egg is conical in shape, yellowish in colour, transversely striated, about 1·25 mm. long, and attached along one side of the broad end by two parallel flanges which encircle the hair. At the free end the egg is provided with an operculum rather obliquely placed.

The eggs hatch out according to Bracy Clark in 25 to 30 days, according to Osborn most readily about the 14th day, and lose their vitality in 28 to 40 days, according to Bedford in 7 to 13 days, in the latter time most commonly. It would appear that for the purpose of hatching out two factors are necessary: (1) moisture and (2) friction such as would be caused by the horse's tongue. The operculum is raised up and the larva leaves by this opening in the egg. It has been suggested that since the horse does not ordinarily lick or nibble itself that the eggs or the larvæ within them cause a tickling sensation sufficient to induce the horse to do so. The larvæ when hatched out become attached to the lips or tongue during licking or nibbling. In this way they reach the mouth, are swallowed, and so find their way into the stomach.

When hatched the larvæ are white, long, and fusiform, with thirteen segments carrying dark spines on the anterior margin, save on the 11th, 12th, and 13th.

On reaching the stomach each larva becomes attached by two hooks at its buccal end to the mucous membrane, which they penetrate. The parts where larvae are most commonly found are the cardiac portion, occasionally the pylorus and duodenum. Immense numbers are sometimes found in the cardiac portion of the stomach. At the posterior end of the larva are two spiracles or breathing-pores. The larvæ or bots undergo three moults. When fully developed they are 16 to 20 mm. long, yellow or reddish yellow, conical in shape, being narrow at attached portion, more convex on dorsal than ventral aspect, and presenting eleven segments carrying backwardly directed spines, brown in colour, black at apex, arranged in two rows on their anterior borders, complete on the 2nd to 8th, incomplete on dorsal surface of the 9th, and almost absent on the 10th segment, with breathing-pores on the posterior aspect of the terminal segment.

They live in the stomach for 10 or 11 months.

In the spring and early summer they become detached and are passed out of the alimentary canal with the fæces. On the ground they burrow into the loose earth and pupate. The outer skin hardens, forming the pupa case, and becomes dark brown. After 18 to 25 days the adult fly emerges from the pupa case and the life-history is again repeated.

Gastrophilus hæmorrhoidalis, Linné (1761), the red-tailed bot-fly, is found in Europe and North America. The adult insect is about 9 to 11 mm. long, very hairy, and of a brownish-black colour. The dorsal surface of the thorax carries olive-grey hairs in front and behind a black transverse band; the abdomen is whitish in front, black in its median part, and orange red at the posterior end.

The eggs of this fly are black and conical and are laid on the long hairs of the horse's lip. The larvæ hatch out more quickly than those of *Gastrophilus intestinalis* and are taken into the mouth with the food or by the tongue when licking the lip, and thence pass to the stomach, where they become attached usually to the cuticular portion, more rarely the fundus and duodenum; exceptionally they may be found in the pharynx. The fully grown larvæ differ from those of the *Gastrophilus intestinalis* in being smaller (from 12 to 15 mm.), not so stout, of a more or less distinct red colour, and in the number and situation of the spines.

On leaving the stomach they sometimes attach themselves to the rectal mucous membrane, may even pass through the intestines independently of the fæces, and fix themselves to the outer margin of the anus, causing some irritation. According to Osborn, because of this habit and their frequent attachment to the mucous membrane of the pharynx, the right half of the stomach and duodenum, these parasites are the most injurious of the Œstridæ which attack the horse.

Gastrophilus nasalis, Linné (1761) (*Œstrus duodenalis*, Schwab, 1840), popularly known as the chin-fly, is met with throughout Europe, North America, and in Asia, and is very common in South Africa.

The flies vary from 10 to 12 mm. in length, are very hairy, coloured reddish brown on the head, thorax black covered with reddish-yellow hairs, and abdomen black covered with fine hairs which vary in colour, white on second segment and black on the third in the female and either black or orange in the male. Wings unspotted.

The life-history differs from that of the *Gastrophilus intestinalis* in the following points: the eggs are laid on the hairs of the lower lip and submaxillary space and mandibles. They hatch out in a few days independently of moisture or friction. They then probably drop on to the food with which they are picked up by the horse, and they become attached usually in clusters to the mucous membrane of the duodenum near the pylorus. The larvæ when fully developed vary from 13 to 16 mm. in length, are blood-red in colour, and carry only one row of spines—on the anterior border of the segments excepting the last.

Gastrophilus pecorum, Fabr. (1794), is found throughout Europe and is very common in certain parts of South Africa.

The adult fly is about the same size as the *Gastrophilus intestinalis*, head brown with sparse covering of yellow hairs, thorax dark brown or black clothed with golden hairs, abdomen black sparsely covered with yellow hairs save in the posterior portion in which the hairs are sometimes black. Wings are slightly fuscous with a dark transverse band.

The life-history differs from that of *Gastrophilus intestinalis* in the following particulars: The larvæ are found in the right and left portions of the stomach, sometimes the pharynx, and occasionally the duodenum. When mature they are reddish in colour and spines are absent from the middle of the dorsal surface of the 6th to the 8th segments.

Prevention.—The measures which are adopted to interrupt the life-history of the fly and eradicate bots in equines consist in preventing egg-laying or destroying the eggs when they are laid.

In the former case one has to remember that the flies are most active in bright sunshine, and therefore if equines are kept in the house during the daytime in the fly season and let out to grass at night egg-laying will be to a large extent prevented. This measure of course cannot always be carried out.

In the latter case the coats of the equines must be examined daily, or at least every few days, during the fly season for the eggs which may have been deposited. When found they should be destroyed, and this can be done in a variety of ways: (1) by singeing the hairs; (2) by scraping the eggs off with a knife and burning; or (3) by touching the patch of hairs carrying the eggs lightly with a cloth saturated with benzene, petrol, or kerosene.

Curative Treatment.—See page 313.

The Bot-Fly of Sheep, Sheep Gad Fly or Sheep Nostril Fly (Œstrus ovis, Linné, 1761), belongs to the same order and family as the horse bot-fly and the ox warble fly. The adult fly is about 10 to 12 mm. long, slightly larger than the ordinary house-fly, greyish yellow in colour, and hairy. The dorsal aspect of the thorax is greyish brown with small black tubercles each carrying a fine hair, the abdomen yellowish white with black hairs, the wings are transparent and large. The mouth parts are not developed and the fly does not feed.

This fly appears in the warm weather during summer and autumn in the neighbourhood of sheepfolds and places frequented by sheep. The females are active only when laying their young. These they deposit on the inner side of the nostrils of sheep and occasionally goats. Young sheep are most commonly attacked. This attack upon the sheep causes considerable annoyance; the sheep affected often snort and rush about with the nose down, and rub their nose on the ground or with their fore feet. The young larvæ which are laid crawl up the nasal chambers and migrate to the frontal sinuses, even as far as the horn cores, where they may be found attached to the mucous membrane in various stages of development. When mature the maggot, or head maggot as it is called, is white in colour, about 25 mm. long, flat on the ventral aspect, convex above, presenting 12 segments, the under-aspect of which carries small brownish spines. It is provided with two mandibles in front for attachment, and at its posterior extremity with two spiracles.

The maggots become mature in about 8 to 10 months. In June and July they become detached from the mucous membrane, are discharged by way of the nostrils, and fall to the ground. There they bury themselves in the loose earth, become quiescent, and change into pupæ. From the pupæ the adult flies emerge in from three to six weeks. These larvæ in the frontal sinuses and nasal chambers often cause considerable trouble.

The *Œstrus ovis* has been recognized throughout a large portion of the globe and is not uncommon in Europe, Asia, and America. It has been stated that the larvæ have been found not only in sheep and goats but even in young boys herding sheep, and sheep-dogs in occasional cases.

Prevention.—In order to prevent the attacks of these flies and exterminate them various methods have been suggested and adopted, but they are not absolutely efficacious. They are chiefly directed to interference with egg-laying. Where it is possible and the flies are very prevalent, the sheep should be kept away from the infested pastures during the fly season. Another measure consists in applying a dab of tar or stinking animal oil to the nose of the sheep to ward off the attacks of the fly. It is sometimes arranged that the sheep's noses are smeared automatically. The troughs for salt lick or food are covered over with boards in which two-inch holes are bored at intervals of six inches. The sides of these holes are smeared with the material, and the sheep in trying to pick up the lick or food from the trough through the holes in the cover smear the oil or tar over their nostrils (see page 398).

J. F. C.

Blood-sucking Flies

Blood-sucking flies all belong to the order Diptera. They are provided with two wings, the posterior pair being reduced to little knobs, and their mouth parts are adapted for piercing the skin and sucking blood. They cause annoyance to animals by their attacks; sometimes the bites cause painful swellings, but their chief importance lies in the part which many of them play in the transmission of various diseases. Among the more important blood-sucking flies are mosquitoes, gnats, midges, black flies, breeze flies, stable flies, and the tsetse flies.

Mosquitoes and Gnats.—Mosquitoes and gnats belong to the family of Culicidæ and comprise over 600 species. They are often mistaken for midges and look like small "Daddy-long-legs." Their bodies and limbs are long and narrow and their wings large, narrow, and scaly along the veins and borders. The two types are *Anopheles* and *Culex*. Common species are *Anopheles maculipennis*, Mergin, 1818, the spotted mosquito, which is about 7·5 to 10 mm. long; and *Culex pipiens*, Linné, 1758, the common gnat, which is about 4·5 to 6 mm. long. Other species include *Anopheles bifurcata*, *Myzomyia superpicta*, *Culex fatigans*, *Stegomyia colopus*, *Theobaldia nemorosa.* Mosquitoes are found in almost all parts of the world, but they are less numerous in temperate climates and most active in warm weather at sunset. The females only are blood-suckers and attack both man and animals. These insects are of greatest interest to students of Human Preventive Medicine on account of the transference by some species of malaria, yellow fever, and filariasis. In the lower animals the anophelina are capable of transmitting the larvæ of *Filaria immitis* of the dog, and probably some mosquitoes are the vectors of South African horse-sickness and blue-tongue of sheep. The great advances which have been made in stamping out the human diseases mentioned have been due to the knowledge which has been acquired regarding the life-history of the transmitting flies.

The mosquitoes lay their eggs on the surface of various collections of standing water, pools, puddles, ditches, swamps, etc. After a few days larvæ hatch out (wrigglers), which are very active in the water and require to come to the surface at intervals to breathe. After a fortnight or so they moult and become pupæ (tumblers), which also breathe in the same way. From these in a few days the adult mosquitoes emerge and then leave the water. The females hibernate in the winter months in sheltered spots. The preventive measures which have proved most effective in dealing with these pests are directed towards the removal of the breeding-places and destruction of the larvæ. Where possible all stagnant water is drained off. In the event of this being impracticable, the water is covered with a thin layer of paraffin or kerosene, and by this means the larvæ and pupæ asphyxiated. Against the admission of mosquitoes to apartments, elaborate arrangements of screens are used.

Midges or Chironomidæ.—Midges or *Chironomidæ* are distinguished from mosquitoes by their small heads, the absence of scales on the wings or body, and the different arrangement of veins on the wings. They are often found in the neighbourhood of streams and woods. Many of them are very annoying to man and animals on account of the painful bites they cause. They have not been shown to be concerned in the transmission of disease.

Black Flies, Sand Flies, or Buffalo Gnats.—Black flies, sand flies, or buffalo gnats belong to the family *Simulidæ*. They are small dark-coloured flies from 1 to 4 mm. long, stoutly built, with a humped thorax and broad large iridescent wings. The females only are blood-suckers. They are world-wide in their distribution, and swarm in damp places in the neighbourhood of marshes and mountain streams. They lay their eggs in marshy places and the larvæ are aquatic. These flies attack both man and animals and produce most painful bites. Of these *Simulium reptans*, *Simulium maculatum*, and *S. cinereum* are common in Europe; *Simulium damnosum* is the Jinga fly of the Nile; *Simulium columbatezense*, Schoub, Kolumbatz fly, is found along the Danube; and *Simulium purnarum*, Rily, is widely distributed in America. The two last species are responsible for considerable damage to stock and dangerous even to man. They are most active at sunset, and sometimes appear in thick clouds and attack the animals in swarms. The animals become restless, even frantic, fling themselves on the ground, groan and bellow. They become covered with flies, which even enter the natural openings. Painful swellings form all over the body, especially on the mucous membrane of the nose, mouth, eyes, and vagina. The animals may die in a few hours from asphyxia due to swelling of the mucous membrane of the respiratory passages. Those lightly afflicted are ill for several days and present small hard painful swellings on the skin and visible mucous membranes which remain for a few weeks.

The treatment adopted consists in the application of cold compresses and cooling lotions or soothing liniments, lead lotion, Carron oil, or carbolized oil to the affected areas, and scarification of the swollen mucous membranes. In dyspnœa tracheotomy may be necessary.

Gad Flies, Horse Flies, or Breeze Flies (Tabanidæ).—These are large flies from 10 to 25 mm. long, with large head and broad, rather flattened body, short antennæ, and large wings with characteristic venation. The females only are blood-sucking, and can penetrate the thickest skin with their proboscis. They are active in their flight in warm sunshine, cause considerable annoyance to stock, and may prevent them from thriving. Some species have been suspected of transmitting various forms ot trypanosomiasis, such as Surra, El Debab, Mal de Caderas, and even infectious anæmia of horses. The females deposit their eggs in masses on leaves, stems of plants, rushes in marshy places, and the larvæ live in water or damp earth. The pupæ appear close to the surface, and the adult flies emerge after a period of more than a year. A few of the more common species are the following:

Tabanus autumnalis, Linné, autumn breeze fly, is 18 to 20 mm. long, of blackish tint. Its thorax is covered with greyish hairs and is marked by four longitudinal brown bands. The abdomen presents three rows of white spots, and the outer border of the wings is brownish. It is common in Europe, and attacks for preference the large species of animals, horses and cattle. *Tabanus nemoralis*, Meigen, is 16 mm. long, thorax brown, presents four longitudinal bands, abdomen brown-grey, and each of the rings is marked by four triangular spots. This species is Algerian, and attacks dromedaries, in which it is regarded by Sargent as transmitting El Debab.

Tabanus tomentosus, Macquart, is a slightly smaller fly than the preceding, which also attacks dromedaries in Algeria and may transmit El Debab.

Tabanus tropicus is noted in India, where it may be a vector of Surra. *Tabanus lineola* is another species of a like nature in India.

Tabanus morio, Lat., the black breeze fly, is of a shining black colour. The face, nearly nude in the male, is hairy in the female. It is found throughout Europe, and attacks man and animals.

Tabanus bovinus, Linné, the ox breeze fly, is about 27 mm. long and of a brown colour, the thorax is covered with yellow hairs and is marked by longitudinal bands, and each segment of the abdomen on the middle of its dorsal surface presents a white triangular spot. It is common in Europe, and attacks chiefly horses and cattle. Other species include *T. rusticus*, *T. oomius*, *T. fulvus*, *T. albipes*, *T. albifacies* in North Africa; *T. atratus*, *T. costalis*, *T. annulatus*.

Hœmatopota pluvialis, Linné, cleg, brump, or small rain breeze fly, is a small breeze fly about 10 mm. long, with rather long narrow abdomen, four pale bands on the thorax, greenish eyes with reddish streaks, and greyish-brown wings. It is common in Europe and elsewhere in the summer and autumn, and attacks both man and animals.

Chrysops cœcutiens, Linné, the small blinding breeze fly, is about 9 mm., with golden-green eyes with three oculi, the antennæ are long, the wings are wide apart, and the abdomen flattened. It attacks the skin round the eyes.

No efficacious method has yet been adopted to ward off these flies or destroy them. Porchinski recommends the application of a thick layer of kerosene over pools to which the breeze flies come to drink. This attracts the flies and the kerosene destroys them.

Horn-flies (family, Muscidæ).—The most important of these flies is *Hœmatobia* (*Siphona*) *serrata*, Robineau-Desvoidy, or Texas fly. It is met with in France and in North America, and is a very troublesome cattle pest. It is about the size of the common house-fly, 4 mm. long; it is grey, with brown palps which loosely ensheath the proboscis. It is similar in appearance to the *Stomoxys*. In the countries where it is common it appears in large numbers in the summer and autumn and attacks cattle most frequently, sometimes horses, and sucks blood. In cattle these flies often rest in clusters round the base of the horns, hence the name horn-fly. They also attack the back, flank, and upper portions of the limbs. They cause great irritation and annoyance, and the cattle lose condition and the milk supply of cows is greatly reduced. It has been suspected that this fly may also transmit anthrax. The horn-fly lays its eggs chiefly in freshly passed cow manure. The eggs hatch out in the summer in twenty-four hours. The larvæ burrow beneath the surface and after three or four days become transformed into pupæ. After six to eight days the adult fly emerges. The effectual disposal or dispersal of the fæces may therefore be useful in preventing the multiplication of the fly, but it is impracticable on large farms. The application of lime to the dung is also useful.

Other species include *Siphona stimulans* and *Liperosia exigua*, found in Java.

Stomoxys or Stable Flies.—*Stomoxys* belong to the same family (Muscidæ) as the house-fly, for which it is commonly mistaken and from which it is readily distinguished by its strong projecting proboscis.

Stomoxys calcitrans, Geoff, is a very common species of this genus. It is about 6 mm. long, brownish grey in colour with a greenish-yellow sheen. The wings at rest are widely spread apart at the tip. The proboscis protrudes like a bayonet at the front of the head. The fly is very common throughout the world, and is frequently seen in stables, byres, and houses. Other species include *Stomoxys nigra*, *S. nebulosa*, *S. glauca*, *S. sitiens*, *S. tæniata*, *S. concolar*. The life-history of the *Stomoxys* is very much the same as for the house-fly. It lays its eggs in fresh manure, particularly horse manure, and moist and decomposing vegetable matter. From the eggs larvæ hatch out; after a time pupæ form, from which the adult fly emerges in a month or so. According to Rouband, the larvæ are destroyed by a temperature above 115° F., and this temperature is exceeded in the centre of a highly packed fresh manure heap. He therefore suggests that fresh manure should always be buried in the centre of the manure heap. The frequent removal of manure from the neighbourhood of buildings will also tend to reduce the number of these flies which enter. The adult fly is very active in the summer and autumn and attacks both man and beast. It is often observed in clusters on the limbs and bodies of cattle and horses. It easily penetrates the skin with its proboscis and causes considerable annoyance, interfering with the condition of the animal. It may even inoculate various specific diseases, such as Surra or Mal de Caderas, and even anthrax and infectious anæmia of horses.

Tsetse Flies, or *Glossina*, also belong to the same family as the house-flies. They have only a regional distribution and are confined solely to certain parts of Africa, especially Central Africa. The tracts of country in which they are common are often called the tsetse belts.

These flies are all intermittent blood-sucking insects, and both males and females are equally culpable. They are active during the day and clear nights; they are very rapid in their flight, producing a buzzing sound and flying in a straight line. They are most abundant along heavily wooded rivers and streams and in the bush where large game are found. The flies are about the same size as, or slightly larger than, house-flies, but their proboscis is long and adapted for piercing the skin and sucking blood. The maxillary palps are as long as the proboscis, for which they form a complete sheath. The wings are large and membranous, extending well behind the abdomen, and when at rest fold over one another scissor-like. They attack both man and animals and alight very quietly on the skin, causing little disturbance. On piercing the skin they gorge themselves with blood in twenty to thirty seconds; they then leave their host and fly into the bush to rest and digest their food. They are viviparous and deposit their larvæ at the root tangles of banana, mangrove, and other vegetation. There the larvæ pupate in a few hours and the adult flies emerge in six to eight weeks.

The genus *Glossina* includes a number of species, of which the following are the more common: *Glossina morsitans*, West; *G. palpalis*, Rob. Desv.; *G. pallipides*, Austen; *G. longipalpis*, Wied.; *G. tachinoides*, West; and *G. fusca*, Walker.

Glossina palpalis, Rob. Desv., is about 8 to 9 mm. long, of a light brown colour dusted with grey. The antennæ are dusky and the arista carries 18 hairs. The upper aspect of the abdomen is nearly black with a median light brown longitudinal stripe. The halteres are white. This fly is found from the Congo to Senegal wherever there is water.

Glossina longipalpis, Wied., is about 8 to 10 mm. long, light brown in colour, and antennæ with 25 aristal hairs. It is met with in British Central Africa.

Glossina morsitans, West, W., is slightly smaller than *G. longipalpis* and its proboscis is twice as long as the head; the thorax presents four longitudinal black lines and the abdomen carries large black spots on the last four segments. It is widely distributed throughout Africa.

These flies are of immense importance because of the essential part they play in the transmission of many forms of trypanosomiasis in man and animals, including sleeping sickness in man, Nagana, Baleri, and Souma. They appear to act not only as mechanical carriers, but in their bodies the Trypanosomata also undergo certain cyclical changes.

The problem of the eradication of these flies or the protection of man and animals against their attack is not yet solved. On account of their preference for bush country, and in some cases for water, it is recommended that the jungle or bush should be cleared from the neighbourhood of rivers and for a considerable distance, at least 400 yards, around habitations. The destruction of big game has also been proposed in order to starve the flies, which live only on the blood which they suck from their hosts.

Hippoboscidæ include the Sheep Ked (*Melophagus ovinus*), already noted; *Hippobosca equina*, Linné, the louse fly or spider fly; *Lipoptena depressa*, Say., of the deer; and *Hippobosca rufipes*.

Hippobosca equina is about 8 mm. long. Its head is yellow and distinct, its thorax brown, and abdomen yellowish brown. Its wings are oblong and rounded. This fly often attacks the horse in the summer; it is difficult to dislodge and rapidly runs over the skin. The horse when attacked becomes very restless, often kicks and plunges.

Measures for warding off the Attacks of Flies.—Natural weapons of offence and defence with which animals are provided, such as the mane of horses, long tails, panniculus, are constantly made use of, but they do not completely avoid attacks or dislodge the flies. Where flies are causing considerable trouble, ear-caps, tassels, nets, and branches of walnut trees attached to the gear in horses tend to dislodge the pests or prevent them from alighting upon the skin. The use of a light sheet for horses in the stable in summer to some extent protects the body.

Various protective dressings have been recommended, but their action is very ephemeral and they require to be repeated at intervals of one or two days. They include paraffin or kerosene emulsion, creolin or wood creosote in whale or other bland oil, decoction of walnut leaves in vinegar, castor oil, fish oil. Castor oil was found by Cross to be the most effective and lasting dressing for camels against Tabanidæ and *Stomoxys*. The burning of fires producing dense smoke in the fields or houses has been tried and found useful to ward off the attacks of flies in stables or even in the open. Naturally, of course, the use of this measure has very distinct limitations. The keeping of animals in the house in the daytime in warm weather is of advantage in some cases in preventing the attacks of flies which are active only in warm sunshine. To prevent flies from entering buildings the light from the windows may be subdued, and an elaborate system of netting is adopted for windows and doors. When flies are numerous they may be destroyed by fumigation with sulphur when the house has been vacated, or fly papers or traps or formic aldehyde used. A 3 per cent solution of formaline in water or milk sweetened with sugar in shallow vessels attracts many of the flies, which are killed on drinking the fluid.

Bees and Wasps.—Bees and wasps belong to the order of Hymenoptera. They are not of great importance in veterinary practice and seldom cause trouble. They are seldom aggressive, and usually only sting in self-defence. The stinging apparatus is carried only by the females and neuters. It consists of a hard director placed at the posterior extremity of the body, which pierces the skin and allows the entrance of the venom, secreted by a special set of glands, into the tissue. Animals are sometimes attacked by honey bees (*Apis mellifica*) when they go near the bee-hives or when the bees are swarming. Horses, cattle, and dogs may also be stung by the bumble-bee (*Bombus terrestris*) when their nests in the ground are disturbed, as in ploughing. *Vespa vulgaris* (the common wasp) occasionally stings dogs when the latter are too inquisitive and nose round these insects. The director is often left in the bodies of these animals after stinging. The sting causes some local painful swelling, which is very irritating and remains for a day or two. If the stings have been numerous the swellings are numerous and may be large. Should they have been inflicted in considerable numbers there is considerable excitement, and later prostration, frequent pulse, diarrhœa, hæmoglobinuria, and jaundice. Even death has been recorded in the lower animals as due to this cause. If the head, mouth, throat, and nose are affected considerable dyspnœa may result from the swelling.

Treatment consists in the application of weak ammonia solution or a solution of bicarbonate of soda to neutralize the formic acid which composes the sting. The application of a cooling lotion such as lead lotion is useful. When there is much dyspnœa it may be necessary to perform tracheotomy. Stimulants are useful where prostration is noted.

J. F. C.

SNAKE-BITE

In the British Isles, and throughout the greater part of North-Western Europe, this subject is of theoretical rather than of practical interest, except in certain localized districts particularly infested with vipers, but it attains very considerable importance in tropical and sub-tropical countries.

In such warmer countries, snake-bite may be the actual cause of many deaths and cases of serious illness among domesticated animals; and, on the other hand, it is apt to be assumed as the cause of all suddenly arising local swellings; cases of snake-bite may therefore be confused with cases of such a disease as anthrax.

The relative frequency with which cases are encountered in the various species of domesticated animals will naturally depend to some extent on the relative numbers of the animals present, and on the conditions under which they are kept, and will therefore show some variation in different countries and districts, but, generally speaking, the dog is the species most frequently affected, and sporting dogs probably suffer to the greatest extent. Cases of snake-bite are commoner in cattle than in horses, and sheep are not infrequently attacked. Cats are very much less frequently affected than dogs, although some cats are expert and keen destroyers of snakes; the cat probably owes its relative safety to its rapidity of movement combined with great caution.

Pigs are also quite rarely affected, and devour snakes very readily, so that it is said to be a practice in some parts of the Mississippi area to turn pigs on to low marshy ground known to be badly infested with young rattle-snakes. The pig is certainly more resistant to snake venom than a number of other species, such as dogs, cats, or donkeys, but there is no true immunity, and the rarity of cases of snake-bite in pigs is probably due principally to the mechanical protection afforded by the thick subcutaneous layer of adipose tissue.

It is of interest to note that the Indian mongoose is very resistant to the action of venom, but here again it is rather its rapidity of movement which enables this animal to attack and kill venomous snakes almost with impunity.

There are also other natural enemies of snakes, such as the hedgehog, the Secretary-bird of South Africa, and certain herons in South America.

Classification.—It is not proposed here to give more than a very rough outline of this subject, but a certain superficial knowledge of the classification of snakes is always of interest, and may be of considerable utility, to the practitioner in warm climates.

In the first place, it cannot be too strongly emphasized that size, colour, and skin markings are by no means always reliable guides to the nature of a snake; in many cases observations restricted to these details lead to most erroneous ideas. As regards size, it is obvious that all snakes are at first of very small size, and only attain their maximum length after a considerable period of more or less steady growth. With respect to colour, individuals of the same species may show very great differences, and considerable changes may be observed during the life of one individual; similarly, skin markings may vary considerably in different individuals of one species, or even in one individual at different periods of its life.

In some cases certain peculiarities of form, size, colour, or habit are so striking that there is little or no chance of their passing unobserved or being misinterpreted. Thus a snake which, when enraged, flattens the ribs in the cervical region so as to form a distinct "hood," may safely be identified as belonging either to the genus *Naia* (the Cobra genus) or to the genus

Sepedon (African Ringhals or spitting snake, closely allied to the Cobras), and is to be regarded as venomous. Again the puff adder of Africa (and Arabia) is easily recognizable on account of its distinctive size, form, colour, and skin markings, as in this case the variations observed are not sufficient to mask the identity of the snake to the observer of ordinary African experience. On the other hand, a cobra which fails, while under observation, to expand the hood may not be recognized as such, while the harmless mole snake is very frequently mistaken for a cobra; similarly, the perfectly harmless egg-eating snake (*Dasypeltis scabra*) is very often thought to be a night adder, as there is some resemblance in size, colour, and markings. Such confusion may lead to much unnecessary alarm amongst human beings, or possibly in some cases to a dangerous lack of precaution; on the other hand, from this cause some line of treatment adopted for a bite may gain a totally undeserved reputation.

It is very desirable for any veterinary practitioner residing in a snake-infested district to learn to recognize most of the species present, and this he can do by a little careful study of killed specimens, but it is important from the first to distinguish between venomous and non-venomous snakes. For this purpose it is only necessary to examine the dentition of the upper jaw.

Ordinary **non-venomous snakes** show two long rows of teeth on each side of the upper jaw, an outer maxillary and an inner palatine row, numbering about 35 and 20 teeth respectively. These teeth, which are small and curved backwards, are all solid and show no very striking variations in size.

In the upper jaw of a **venomous snake,** such as a cobra or an adder, the palatine teeth are very similar, but the outer maxillary row is represented on each side by a single tooth or fang, which relatively is very large, is situated near the anterior extremity of the maxilla, and which is more or less covered by a loose fold of mucous membrane. This membranous fold contains a series of very small fangs in different stages of development, so that a fang which is broken off or shed is replaced by the first (and furthest developed) of the replacing fangs. The fang is deeply grooved in snakes of the cobra type, whereas in vipers or adders the fang, which is very long and sharply pointed, is pierced throughout its length by a central channel. The channel or groove communicates at the base of the tooth with the duct leading from the poison gland, which is situated behind the eyes, and can be violently compressed by contraction of the masseter muscle.

From the foregoing description it will be realized that a bite inflicted by a non-venomous snake will generally produce two double rows of numerous small punctures, whereas that of a venomous snake will cause two single rows of such small punctures, and one relatively large and deep puncture on each side, external to these single rows. These details are sometimes observable in human beings and in animals where the skin is hairless or shaved.

Finally, it may be remarked that there is an **intermediate group of snakes,** known as the Opisthoglypha, in which certain teeth situated towards the centre of the maxillary row are of larger size and grooved. These snakes have generally been held to be only very slightly venomous, but we have been able to show that one member of this group (*Dispholidus typus*, the South African "Boomslang") is, under conditions favourable to the snake, able to inflict bites fatal to horses, mules, baboons, and sheep.

Period of Incubation.—With respect to the period of incubation, symptoms may be shown within five or ten minutes of the bite, while in other cases no evidence of systemic disturbance may be noticeable for some hours, and even for a day or longer.

Local swelling, in those cases in which a swelling forms, may also be noticeable within five or ten minutes of the infliction of the bite, or it may often take some hours to become sufficiently large to attract notice.

In considering the effects produced on animals by snake venom, it is most important to realize that the nature and severity of the symptoms depend not only on the species of snake concerned, but also on the dose of venom actually injected into the wound and absorbed into the circulation, and this dose is liable to great variations under the influence of a number of different factors.

In the first place, the age and size of a snake will naturally bear some relation to the size of the poison glands and to the amount of venom which can be secreted and accumulated. In this connection, however, we have shown that young "Ringhals" snakes, when less than twenty-four hours old, are able to inflict a bite fatal to the guinea-pig, while a Ringhals aged one month, and rather less than 6 inches in length, was able to kill a large Irish terrier, so that a young and small snake may be able to secrete more than a lethal dose of venom.

More important factors are the time which has elapsed since the last occasion on which a bite was inflicted or prey was ingested, and the nature and depth of the actual lesions produced by the fangs in biting. The bite of a snake is naturally less dangerous very soon after the reptile has bitten some other object, and if the bite is inflicted through clothing, or thick hair or wool, or if the fangs are not driven directly into the flesh, a large proportion of the venom may fail to be injected subcutaneously, and therefore will not exercise any action on the bitten organism.

There are certain snakes (*Sepedon hœmachates*, the Ringhals of Africa) which, in addition to biting, have also the ability to project venom into the eyes of their prey or enemies. This is brought about by violent contraction of the masseter muscles attached to the capsules of the poison glands, the head being raised and the mouth opened, and these snakes show great skill in directing the flow of venom at the opponent's eyes. In this way venom can be projected for a distance of at least 6 feet.

Apart from the variations due to the above-mentioned causes, it is necessary to recognize two distinct groups of cases arising from the action of colubrine (cobra type) and viperine (adder type) venoms respectively.

There are a number of symptoms common to both types of cases, and in some cases it might be difficult to determine, from clinical symptoms alone, the nature of the venom injected.

Generally speaking, however, colubrine venom exerts its chief and most noticeable action on the nervous system, and particularly on the mechanism governing respiratory movements, while in cases due to viperine venom the local action is very well marked. It is therefore convenient to describe the two types of cases separately.

A. *Symptoms shown by Animals bitten by the Cobra Type of Snake.*—The general course may be summarized thus :

(*a*) In the majority of cases there is a period of excitement and restlessness, which may last for a few minutes only, or may persist for an hour or longer, especially in those cases in which local pain is noticeable.

(*b*) The preliminary excitement may be succeeded by a period during which the animal appears normal.

(*c*) Later, in the majority of cases (but not in all), there are symptoms of nervous excitement. Within one to two hours (or in larger animals within four or five hours) of the bite the animal may show convulsive muscular contractions, and may within a short period die of asphyxia. This is seen particularly in sheep and dogs, but may occur in the larger domestic species.

(*d*) Animals not succumbing early from asphyxia generally develop symptoms of general depression, sometimes interrupted by periods of restlessness and excitement. As a rule, in cases surviving for a long time there is a final stage of dulness and depression, which persists until death occurs, or until there are general signs of recovery.

These various phases occur in the majority of cases, but the duration and intensity of any one stage is liable to very wide variation ; thus in some cases of cobra-bite symptoms of nervous excitement are very well marked, while in other cases such symptoms are almost or completely lacking, and symptoms of depression predominate.

In many cases no evidence of pain is shown, while in other cases many symptoms of local pain are observed. Such symptoms include extreme restlessness, repeatedly lying down and rising again, sweating, hurried respiration, and a frequent hard pulse. There may also be symptoms of local pain especially associated with the particular area involved, such as lameness, abnormal gait or posture.

Symptoms of nervous excitement, apart from manifestations of pain, include restless movements of the head, limbs, or tail, restless wandering about the box or stall, grinding of the teeth, salivation, frequent movements of deglutition, and frequent defæcation and micturition. Animals may also show such signs of cerebral disturbance as licking walls, biting the air, and knocking the head against the wall or manger. During this stage of excitability the animal generally shows frequent deep respirations and a frequent fast pulse.

Quivering of muscles, or even strong spasmodic contractions, may be seen, and in each case the process may be local or more or less general. Immediately before death there may be asphyxial convulsions.

A number of the above-mentioned symptoms may arise from pain, and in some cases it is difficult to determine whether the symptoms are due to pain or not.

Under the heading of symptoms of " Nervous Depression " one may include a group of symptoms varying from slight dulness to paresis, either local or more or less general, and even paralysis. In the dull stage the animal is inclined to remain for long periods in one position ; the head hangs, the ears droop, and frequently the eyes are closed. Later the animal may go down, or it may rest against a wall. Co-ordination of movement may become impaired, resulting in a staggering gait, and loss of control over particular parts may be noticeable. Thus the lower lip may fall away from the teeth, and the lips are then unable to close on any food, although an effort may be made ; the anus may be relaxed, and in equines the animal may be unable to protrude the penis in micturition, or to retract it when protruded. Animals in this stage sometimes show well-marked salivation.

The corneal reflex is generally obtained until very shortly before death, but where symptoms of paresis or inco-ordination are shown, the reflex may be delayed or slow.

General dulness and depression are usually seen in animals that eventually recover, although such a stage also occurs in many fatal cases.

Animals showing symptoms of inco-ordination, loss of control, or paralysis in our own cases have always later become comatose and died.

The temperature is not noticeably affected.

In rapidly fatal cases no local swelling is seen, but a noticeable swelling is present in many other cases, and particularly in cases which survive for many hours, or even days, and in those which end in recovery. Consequently, such swellings are more common in the larger animals and in those smaller animals which have received less than the lethal dose of venom. The swellings vary greatly in size and extent, and also in character. In some cases the local lesion is soft and insensitive, and it is then frequently pendulous, while in other cases it is hard or tense, warm to the touch, and very sensitive to manipulation ; the larger swellings are generally soft and insensitive. These swellings show a great tendency to gravitation, and may therefore be most prominent in regions situated at some considerable distance from the lesions inflicted by the fangs. In cases of recovery the swelling may persist for a week or longer, but it has usually disappeared at the end of two weeks.

Death generally occurs through stoppage of the respiratory movements, and the heart may continue to beat for three or four minutes after all signs of respiration have ceased.

Duration.—In equines death frequently occurs on the second day after the bite, and the most rapidly fatal case we have seen was that of a mule (bitten by the yellow or Cape cobra), which died in slightly over five hours.

Sheep and dogs frequently succumb within two hours, and even in considerably less than one hour, but death may not occur until the tenth hour or later.

B. *Symptoms due to the Bite of the Adder Type of Snake.*—The general course of the attack shows some resemblance to that seen as a result of a cobra-bite, the following stages being noticeable :

(*a*) A period of restlessness and excitement, which appears to be due chiefly, if not entirely, to local pain.

(*b*) In some cases the first stage is succeeded by a period during which the animal shows little abnormality, apart from the presence of a local lesion, which is generally large, and increases rapidly in size.

In most cases, however, signs of pain are very noticeable at frequent intervals or almost continuously.

(*c*) In a few cases dyspnœa sets in early—after an hour or less in dogs and sheep, or a few hours in the larger animals. In such cases the animal dies within a short period, after showing muscular tremors and contractions, or even marked convulsions, followed quickly by depression, coma, and death.

(*d*) Animals surviving for longer periods pass through a final, and often prolonged, stage of depression, which may end in recovery or death.

These cases generally differ from the majority of those due to the cobra type of venom, in that a local swelling is a prominent feature, and that there are generally obvious signs of acute local pain.

Livid discoloration of the skin around the punctures caused by the fangs is common, although it may not be easily perceived, on account of a thick growth of hair and cutaneous pigmentation ; such a lesion may also occur after cobra-bites, but with viperine venom the effect is more marked.

In animals which succumb rapidly, as a result of the early onset of dyspnœa, the local swelling may not be very noticeable, but in the great majority of cases it is large and prominent, and it may be enormous. Some swelling is usually noticeable within a quarter of an hour, in the form of a more or less circular elevated area of skin, over and around the punctures. At first the swelling is rather soft, but it rapidly becomes tense, warm to the touch, and very sensitive to palpation. In most cases it increases rapidly in size and shows a well-marked tendency to gravitation. In cases ending in recovery the swelling later becomes softer and less sensitive, and it decreases in size rather slowly.

Local pain is generally present in a very much greater degree than is seen in cases due to the bite of the cobra type of snake. The usual symptoms are great restlessness and excitement, grinding of the teeth, profuse sweating, frequent micturition and defæcation, hurried respiration, and a frequent hard pulse. Owing to the usual occurrence of much local pain and swelling, there are generally pronounced symptoms associated with the particular area affected by the bite ; thus if one of the limbs has been bitten, there is generally well-marked lameness, and the affected limb may be carried or rested, or it may be moved restlessly from time to time.

We have never observed any signs of inco-ordination, paresis, or paralysis as a result of a viperine bite, nor such cerebral symptoms as licking walls or biting at the air.

In those cases in which dyspnœa sets in early and the animal dies of asphyxia there may be twitching of various groups of muscles, and later strong muscular contractions and involuntary defæcation.

After the more painful phase has passed, or during apparent intervals of relief from the pain, the animal is usually dull and depressed, standing for a considerable time in the same position, with hanging head, drooping ears, and frequently with the eyes closed. The pulse becomes infrequent and weak and the respiration laboured.

At a later stage the animal shows increasing weakness, sometimes swaying from side to side, and then lies down; in some cases the neck is stretched and the muzzle rested on the ground.

In fatal cases the weakness increases, the pulse and respiration become progressively weaker and less frequent, and the conjunctival reflex slower and delayed. Finally the animal lies in a comatose condition, insensitive to various stimuli, with stertorous respiration and a weak infrequent pulse until death occurs.

Post-mortem Lesions.—A. *Lesions due to Colubrine Venom* (*Cobra Type*).—There is generally a local lesion, although this may be very small and not associated with any definite swelling. The lesion usually takes the form of an infiltration of the subcutaneous areolar tissue with a yellowish serous liquid, but sometimes it is hæmorrhagic in parts, and exceptionally there may be extensive hæmorrhagic infiltration. The liver generally presents the changes associated with fatty degeneration, and some pulmonary œdema is often to be noted. Petechiæ or ecchymoses are often present under the epicardium and endocardium, and may be observed in the trachea, under the pleura, and in the stomach and intestines; in the intestinal tract there may be hyperæmic areas, swelling of Peyer's patches, or hæmorrhagic streaks and patches. The blood as a rule is not obviously affected, although most colubrine venoms have been shown to possess anti-coagulative and hæmolytic properties *in vitro*.

B. *Lesions due to Viperine Venom.*—On reflecting the skin over the local lesion, livid areas may be seen around the punctures. There is generally an extensive infiltration of the subcutaneous areolar tissue, and the deep fascia, and even the subjacent muscular tissue, are commonly involved. The œdematous liquid is of a deep red colour, and a gelatinous material, also deeply blood-stained, may also be present. The hæmorrhagic area may be surrounded by a zone infiltrated with a clear yellowish liquid. The liver and kidneys generally show signs of fatty degeneration, and petechiæ and ecchymoses are found in similar situations to those mentioned in connection with cobra-bites, but those resulting from viperine bites are generally more numerous and extensive. With regard to the action on the blood, most viper or adder venoms have a considerable coagulative effect, but certain venoms of this group exert a distinct anti-coagulative action *in vitro*, and even *in vivo*.

In discussing the post-mortem lesions, it is of interest to note here the effect of the venom of *Dispholidus typus*, the South African "Boomslang." This species, which belongs to the group of Opisthoglypha, has generally been considered to be more or less harmless to the larger mammals, but we have succeeded in causing this snake to inflict bites fatal to horses, mules, sheep, etc.

The symptoms, apart from some excitement in certain cases, were those of rapidly developing anæmia and exhaustion produced by internal hæmorrhage, occurring after a very long period of incubation.

On post-mortem examination numerous and extensive hæmorrhages were found. In some cases these were all subcutaneous, reflection of the skin revealing a number of well-defined hæmorrhagic patches scattered irregularly about the trunk, limbs, and head and neck. In other cases hæmorrhage occurred into the respiratory passages, the serous cavities, or the gastro-intestinal canal, and smaller hæmorrhages in the depth of solid organs were also seen.

Diagnosis.—During Life.—In practice it is only occasionally that the actual bite is witnessed, as regards horses, cattle, and sheep, these animals generally receiving the bite when out grazing. The infliction of the bite is more often witnessed in dogs, particularly in sporting dogs, but even in these animals the occurrence of snake-bite may only be surmised by the owner.

When the animal is seen very soon after the bite, the state of excitement may be suggestive, and signs of local pain or irritation may lead to the discovery of the punctures inflicted by the fangs; these punctures may then be bleeding.

At a later stage the formation of a local swelling may attract the attention, and the fang punctures may be discovered. In tropical regions where snakes are common the layman is very apt to ascribe all swellings in animals to the effect of snake-bite, but in many of these regions both anthrax and blackquarter are very prevalent.

The swelling in snake-bite has not the distinctive characters found in a case of blackquarter, and in most cases there should be no difficulty in differentiating them.

A swelling due to snake-bite may resemble considerably some of the swellings seen in cases of anthrax, but in the former case the temperature remains more or less normal, and very often there are general symptoms which are not seen in anthrax.

At certain stages of pain and nervous excitement the case may show some resemblance to one of acute spasmodic colic, but the cases associated with the greatest pain, *i.e.* those due to viperine venom, are usually associated with the formation of a prominent local lesion, and there are often obvious signs that the seat of the pain is not abdominal.

In most cases due to colubrine venom the pain is apparently not severe, and there are usually some nervous symptoms not commonly associated with spasmodic colic.

The occurrence of severe pain in a horse, associated with the presence of a warm, tense, and painful swelling, may arouse suspicion of the "colicky" form of anthrax, but in snake-bite, as stated before, the temperature is little, if at all, raised.

Post-mortem.—In making an autopsy in a suspected case of snake-bite, one will naturally pay particular attention to the local lesion, if any be noticeable, and in all such cases one should examine the reflected skin carefully for signs of the punctures made by the fangs.

Apart from the occurrence of cutaneous punctures and the characters of the local œdema (if present), there are usually no very obvious macroscopic changes on which to base a diagnosis. In most cases, however, the history of the case is very helpful, and such diseases as anthrax, if suggested as possible causes, can be excluded by microscopical examination and, if necessary, by determining the absence of the distinctive lesions.

Treatment.—(1) When procurable, an anti-venomous serum undoubtedly affords the most useful means of combating the effects of snake-bite.

An anti-venomous serum can be prepared from horses, which are first inoculated with small doses of cobra venom mixed with 1 per cent hypochlorite of lime, and later with increasing doses of fully active venom. Such a serum protects animals only against the neurotoxin which is the principal active constituent of such a venom as that of a cobra, and it cannot be expected to prevent the local effects of the hæmorrhagin which is the predominant constituent of most viperine venoms.

A special anti-hæmorrhagic serum can be obtained by the hyper-immunization of horses with viperine venom, however, and it is possible to obtain a polyvalent serum meeting both cases. Such serum is very often not available, however, and in many instances its cost will prevent its extensive use for the treatment of animals other than dogs.

(2) Besides a specific serum which is able to exert its influence on venom which has been absorbed, there are certain chemical substances which are able to destroy venom by direct contact, and which may therefore prove of great value if used within a short time of the infliction of the bite and if introduced sufficiently deeply to come into direct contact with the greater part of the injected venom. Of these substances potassium permanganate and calcium hypochlorite are perhaps the most efficacious.

The best routine procedure in ordinary cases of snake-bite is as follows:

(*a*) As soon as possible, in cases where the situation of the bite renders this practicable, a tight ligature should be applied above the bitten part, and this ligature should be kept tight for half an hour.

(*b*) Two or more incisions, at least 1 centimetre in depth, should be made through or over the fang punctures, and the part should be compressed to encourage the expulsion of venom from the wound.

(*c*) Crystals of potassium permanganate should then be rubbed well into the incisions and fang punctures, or the wound may be freely washed with a freshly prepared 2 per cent solution of calcium hypochlorite, and a few c.c. of this solution may very well be injected into and around the wound with the aid of a hypodermic syringe. The wound may finally be covered with a compress saturated with solution of potassium permanganate or calcium hypochlorite.

(*d*) If available, a dose of serum should then be inoculated; in most cases it is sufficient to inject the serum subcutaneously into a part well provided with loose areolar tissue, but if symptoms of intoxication are already appearing, the inoculation should be intravenous.

It is well to keep the patient warm with rugs, etc., and such stimulants as hot coffee are generally recommended for human patients. Alcohol is not advised when the serum treatment is used, although in other cases it may be beneficial.

A number of agents which were formerly advised, such as ammonia and strychnine, are not now recommended.

In those cases in which venom is projected into the eyes (as sometimes occurs with *Sepedon hæmachates*, the South African "Ringhals," and possibly also with *Naia flava*, the Cape Cobra) it is sufficient to treat the resulting acute conjunctivitis with mild collyria, such as a solution of boracic acid, and to protect the eyes from light.

Literature.—There are a number of records of cases of snake-bite in animals scattered throughout the literature, but for full information with respect to the effects of snake-bite and the actions of venoms the standard work of Calmette should be consulted (*Les Venins*; also English translation by Austen).

Records of a number of experimental cases in domestic animals (bitten by South African snakes) are given by the author in the *Second Report of the Director of Veterinary Research, Union of South Africa*, 1912. W. H. A.

POISONS AND POISONING

The present article on Veterinary Toxicology does not aim at being in any sense comprehensive. All points of a more strictly chemical character are omitted, and only bare reference can be made to the large majority of rarer cases of poisoning amongst animals. Fuller details can be sought in such books as Finlay Dun, *Veterinary Medicines*; Winslow, *Veterinary Materia Medica*; and Lander, *Veterinary Toxicology*.

Definition of a Poison.—It is difficult to find an entirely satisfactory definition of a poison, which, however, in the stricter sense, may be defined as a substance of relatively simple chemical constitution, able to produce in comparatively small doses deleterious or fatal effects. In sub-lethal quantities one expects specific action on the organism affecting some one or more particular groups of cells, so that many poisons in smaller doses act as therapeutic agents, *e.g.* many metal salts and alkaloids.

A poison differs from a bacterial toxine in that it does not give rise to the formation of an anti-body in the blood serum, such anti-bodies being capable of conferring protection against poisoning in a fresh subject. But the phytotoxines, such as ricine, crotine, and abrine, and the zoo-toxines, such as snake-venom, do give rise on sub-lethal injection to anti-bodies, and thus bridge the gap between the relatively simple chemical poison and the complex bacterial toxine. In this connection it is interesting to observe that in distinction to the toxine and bacterial poisons the ordinary poisons do not directly cause rise of temperature.

In a strict sense no agent should be considered a poison unless its action in medicinal doses is marked by specific therapeutic effects. It is desirable to eliminate damage caused by injudicious feeding or by malnutrition from the category of poisoning. Professer Cushny puts it in this way: "Some bodies may be remedies, foods, or poisons according to the quantity ingested and the mode of application."

Although an ordinary poison never produces an anti-body, one must remember that continued dosage may bring about tolerance. This has been observed amongst animals in the arsenical treatment of piroplasmosis, and is further well illustrated by the tolerance acquired by the human subject towards alcohol, tobacco, and morphine.

Absorption, Distribution, Accumulation, and Elimination.—The absorption of a poison depends on the physical nature of the poison and on the channel of absorption. As regards the former condition, the gaseous state represents the most easily absorbable form when the gas enters the lungs. The vapours of prussic acid, carbon monoxide (coal gas), chlorine, and volatile anæsthetics act, as is well known, very rapidly on inhalation. On the other hand, a solid, insoluble in water, acid, or alkali, is in general not poisonous, even although soluble compounds containing the same active ingredient may be so. Thus, whereas the soluble salts of barium are exceedingly dangerous, the insoluble barium sulphate is harmless when taken through the mouth, and among the compounds of lead the effects of the sparingly soluble lead sulphate are much slower than those of the more easily soluble white lead, or the soluble sugar of lead. Coarsely powdered vitreous arsenic may be given in large quantities to dogs without bad effects; the more finely powdered white arsenic is, the more readily is it absorbed, and the maximum effect is noted in the exhibition of white arsenic in alkaline or dilute acid solution. It is clear in all cases that the absorption of a poison takes place by osmosis of a solution of it into the body fluids. Certain very complex bodies such as the toxines are either incapable of undergoing osmotic diffusion or do so with extreme slowness. For this reason snake-venom is stated to be unabsorbed, and therefore to produce no injury on introduction into the intact alimentary canal, whilst ricine of castor seeds is diffusible to a limited extent. The effects of such bodies are therefore incomparably greater when introduced into the system by injection. The vegetable alkaloid curarine, amongst simpler soluble poisons, is to be noted as not very easily absorbed, whilst certain metal radicles, such as magnesium, iron, and bismuth, are not permeable through the living cell when in solution, or at any rate only to an extremely small extent.

In practice the accidental poisoning of animals by gases is exceedingly rare; in the vast majority of cases we have to reckon with absorption in solution through the alimentary tract, and more rarely with absorption through a wound which approximates to the conditions proper to administration by injection.

As regards distribution, it may be taken as established that, apart from corrosives, which produce gross lesions by destroying the living cell, a poison only acts after entrance into the general circulation. In such cases as those of prussic acid and arsenic, for which we have very delicate tests, the distribution in the organs, blood, brain, and muscle may be followed by actual analysis. In so far as chemical methods of detection are lacking in delicacy, a specific physiological effect such as that of a mydriatic alkaloid is often a better test than a chemical reaction. The liver arrests most metals, phosphorus, and many alkaloids on their arrival in the portal system at that organ after absorption, but some substances, such as the salts of sodium and potassium, alcohol, and digitalis, are not so arrested, and obviously the degree to which a poison is held up depends upon the quantity administered. The liver is thus the principal seat of storage of accumulative poison, and it is from the liver in the majority of cases that elimination proceeds, partly by the blood through the kidneys, skin, and milk, but principally by the bile, from which a portion may be reabsorbed and carried back to the liver (Claude Bernard). It is clear that the rate of elimination varies, being most rapid with volatile gaseous poisons and slowest with heavy metals.

Variations in the Effects of Poisons.—These are due to species and idiosyncrasy. The relatively immense dilution in the digestive apparatus of a ruminant, affording greater opportunity for elimination, accounts for the relatively larger *pro rata* dose required to produce ill effects in the ox, sheep, and goat compared with the horse or a carnivore, but the ox is more sensitive to mercury and lead than the horse. The relative degree of development of the nervous system is important, particularly as regards the effects of alkaloids. Thus 2 grains of morphine is used as an anæsthetic for a large dog, which withstands doses very much higher in proportion than those which are injurious to man. This alkaloid, acting as a hypnotic on man and the dog, is an excitant and convulsive on the cat, goat, pig, ox, and horse. Birds withstand large doses of strychnine, and the rabbit is said to be insensitive to atropine. Idiosyncrasy is illustrated in the fact that an ordinary medicinal dose of strychnine is sometimes dangerous to the dog, but in so far as in poisoning a large overdose is usually to be reckoned with, variation due to idiosyncrasy is not so important to toxicology as to therapeutics.

Classification.—Broadly and for practical purposes we classify poisons into corrosive, irritant, and non-irritant nervous poisons.

Corrosives are represented by the strong mineral acids and alkalis, phenols, and very concentrated solutions of many salts. They owe their action to their concentration, so that a poison acting as a corrosive in a concentrated form may have quite a different action when dilute. The effect of a corrosive is marked by actual destruction, by the abstraction of water, by decomposition, and by the solution of fats and proteins in the living cell.

Irritants so modify the cell as to disturb its normal course of metabolism, ultimately causing inflammation. The irritant effect is not limited to special cells of the organism, but is general, and poisons having a wide range of activity are the protoplasmic poisons, such as mercuric chloride, phenol, and prussic acid. The salts of the heavy metals are typical irritants, but nervous effects are often added to irritant effects, leading to the designation of narcoto-irritant applied in practice to so many vegetable poisons. Also with the metals the local irritant effects must be distinguished from the general effects produced after absorption.

Non-irritant Nervous Poisons may be distinguished according to the centre affected. Local effects are not significant and symptoms only set in after absorption.

A fairly complete survey of poisons, arranged according to their physiological effects, is that of Rabuteau :

Hæmatic, acting on blood corpuscles: cyanides, phosphorus, arsenic, alcohol, carbon monoxide, sulphuretted hydrogen.

Hæmatic, acting on plasma: silver.

Neurotic, paralyso-motor : curarine, aconitine, conine, cicutoxin.

Neurotic, spinal : strychnine, cantharidin.

Neurotic, cerebro-spinal : chloroform, ether, morphine.

Muscular : solanaceæ, digitalis, veratrine.

Types of Poisoning.—The three main types of poisoning, acute, sub-acute, and chronic, depend on the dose.

Acute poisoning follows a large dose and manifests intense symptoms, rapid *dénouement* and termination. Irritants cause burning sensations, nausea, vomiting when possible, abdominal pain, diarrhœa, vertigo, collapse ; with nerve poisons, unrest, excitement, delirium, tremors, convulsions, difficulty in breathing, cyanosis, paralysis, and coma.

Sub-acute poisoning, arising from smaller doses, leads to the same train of symptoms less rapidly developed, milder, and more protracted.

Chronic poisoning, consequent on the cumulative effect of repeated small doses, is not common among animals.

Diagnosis.—Sudden illness or death, especially following a meal, dipping, change of situation, or change of diet, is commonly held to indicate poisoning, but obviously this does not constitute full evidence, which is only to be based on the symptoms, history, post-mortem, chemical analysis, and if possible discovery of the concrete source.

Whilst it is impossible, with very rare exceptions, to give general symptoms whereby a case of poisoning may be recognized, the following remarks may be useful.

(*a*) Alimentary symptoms comprise : salivation, foaming, colic, retching, vomiting, purgation (which may be bloody), bloody extravasation of the mouth, tongue, jaws, and fauces.

(*b*) Circulatory symptoms comprise : accelerated or retarded, throbbing, or feeble heartbeat ; hard, imperceptible, or weak, irregular pulse ; cold or hot dry skin ; sweating.

(*c*) Respiratory symptoms comprise: accelerated or retarded, intermittent breathing, with groaning, rattling, or gasping.

(*d*) Motor symptoms comprise: trembling, quivering, cramp, stiffness or twisting of the neck, locking of jaws, epileptiform or convulsive seizures, paralysis of the hind or all the limbs, loss of feeling or great irritability of the skin.

(*e*) Cerebral symptoms comprise: fear, shrinking on disturbance, frenzy and delirium, or dejection, hanging of the head, drowsiness, loss of sensibility, and coma.

(*f*) Other symptoms are contraction or dilatation of the pupil, a fixed anxious look, staring coat, suspension of lactation, repression or incontinence of urine, which may contain blood, albumin, bile, or excreted substances, such as phenol derivatives in carbolic acid poisoning. The mucous membrane of the mouth may show characteristic staining or erosion, and the breath may contain recognizable traces of volatile substances, such as prussic acid.

Treatment.—Although it is rarely possible to diagnose the actual poison at work, the general symptoms as detailed above will afford a guide to the class of poison and suggest certain general remedial measures and precautions.

(1) If poisoning is suspected, where possible change the situation of the animals and the food, especially if the latter be new. Offer only food and water of proved purity. Make an immediate search for suspicious articles, remembering that as these are easily accessible to the animals they should be looked for first in accessible and exposed places. A great deal of time is often lost in a minute and recondite search for a source of poison which lies quite close at hand, *e.g.* a water-cart containing weed-killer. It is useful also to make a search for dangerous or suspicious plants, garden cuttings, and the like. In any case keep the patients quiet and unworried.

(2) Remove any irritant from the skin; if acid, by warm soap or soda solution; if alkali, by vinegar. Clear the stomach where possible, and, unless contra-indicated, by emetic, such as salt, mustard, and water, zinc sulphate, ipecacuanha, or apomorphine hypodermically. Dilute the poison by fluids.

(3) Neutralise the effects. Against irritants and corrosives use demulcents. Relieve abdominal pain by means of opium, morphine, or chloral. Stimulants are indicated against narcotics, alcohol, brandy or whisky, ether hypodermically, strong coffee or tea, inhalations of ammonia. When there is an inclination to drowsiness or coma keep moving, give douches of cold water, apply liniment of ammonia and turpentine in oil to horses and cattle.

Sedatives are indicated against convulsions.

(4) Promote excretion by means of oily purgatives, bearing in mind, however, that oils are contra-indicated in phosphorus poisoning.

Post-mortem Examination.—The appearances should be carefully noted, although unfortunately they are rarely characteristic; the most common observation being that of more or less acute gastro-enteritis. In general, pure alkaloids and other vegetable poisons do not produce irritation, but most mineral poisons and plants do so. A search of the alimentary contents will sometimes reveal the cause. Prussic and carbolic acids, chloroform, alcohol, essential oils, and phosphorus betray their presence by their odours. Fragments of poisonous plants, lead splashes, pieces of paint, and the husks of certain seeds, such as castor, may often be found. It is wise in all events to keep specimens, particularly of vomit or ingesta, for further examination.

Common Causes of Poisoning.—The causes of poisoning amongst animals are very different from those obtaining in the human subject, which generally arise from malice or suicide. For example, poisoning by growing plants is common amongst cattle and horses, but is only found in man in the cases of lunatics and children. Malicious poisoning usually only applies to small animals, dogs, cats, foxes, and poultry, and in that case, in by far the larger number of instances the agent is strychnine given in the form of vermin-killer; less frequently phosphorus vermin pastes and arsenic. The ox is the commonest victim of accidental poisoning, possibly on account of its less discriminate feeding habits as compared with the horse and sheep. Lead preparations, probably on account of their sweet taste, are very often taken by the larger animals, and weed-killer and dip are also very common causes of trouble.

The continual empirical administration of condition powders and the like by cattlemen, horse-keepers, and others, is a practice which cannot be too strongly deprecated. In spite of popular opinion to the contrary, these things do more harm than good. It is the greatest possible mistake to hope to make good on a deficient food by means of medicine.

One of the most remarkable features relating to the poisoning of animals consists in the marked difference noticed in ordinary as against experimental feeding. Whereas animals moving together and feeding together in the ordinary way will partake freely, and indeed seem to eat against one another, thus consuming large doses of poison, the present writer has never succeeded in inducing any isolated experimental animal voluntarily to eat a poisonous dose of any poison. Rather than do so the animal will starve, although it will eat a small dose once. These remarks apply especially to cattle, sheep, pigs, and chickens. It is easier to get a dog to bolt a piece of poisoned meat.

On the whole it is safe to say that the greater number of cases of accidental poisoning of animals are the result of ignorance or gross carelessness, or both, which lead people to leave lying about in exposed situations, dip, weed-killer, paint, disinfectants, and the like.

In the following sections allusion will only be made to the commoner cases of poisoning, and these may be conveniently subdivided into mineral, organic, and plant. In addition to those for which such detail is given, reference will be made for the sake of completeness to cases of the rarer kind, where poisoning has been proved, or alleged to have occurred.

Mineral Poisons

Lead.—*Origin.*—White lead is the commonest and most dangerous cause of lead poisoning. Besides being used as a paint, it is also used in the manufacture of oil-cloth and linoleum. The pigment red lead is also dangerous. Lead sulphate is less dangerous than white lead, but is less frequently used as a paint. The lead paint trade is an exceedingly dangerous one, and white lead substitutes free from lead are coming more into use. Lead splashes, for example, from bullets, have been often fully proved to be dangerous, but the writer has never heard of an authenticated case of poisoning through a solution of lead in soft water. Lead-smelting works emit lead fumes, probably chiefly consisting of particles of lead sulphate in such a minute state of subdivision that it is exceedingly difficult to deprive the fumes of them, and they almost partake of the properties of a gas, being carried to a great distance by air currents (see page 535).

The approximate minimum toxic doses of lead acetate are: ox, 720 grains; horse, 7500 grains; sheep, 450 grains.

Symptoms.—Gastro-enteritis, colic, convulsions, coma, and death are the general effects of the acute poisoning of cattle by lead. Intense abdominal pain, grinding of teeth, nasal discharges, salivation, pallor of mucous membranes, constipation with passage of hard black dung, fœtid breath, ropy urine, blindness, and muscular tremors, are signs. The temperature is nearly normal, or depressed, and the extremities cold. The nervous symptoms, marked by delirium, in which the animal rushes about blindly, often damaging itself against obstacles, often lead to a diagnosis of vegetable poisoning. The blue line on the gums, which is a marked sign of chronic lead poisoning among lead workers, is probably due to the inhalation of lead dust and is rarely or never seen in animals.

The onset of symptoms is often slow. This is well illustrated in a case signalized by Henry Taylor (*Record*, 1915, p. 97), in which a sow, having eaten about two pounds of white lead paint, after suffering gastric derangements looked like recovery after fifteen days, but on the sixteenth the symptoms of violent madness and blindness declared themselves, and the termination was rapidly fatal. This observation points to slow elimination and also indicates that some time may elapse before the lead reaches the nervous centres involved.

Slow lead poisoning such as ensues on the ingestion of bullet splashes may be protracted over such a long period as fifty weeks, and is marked by tucking up of the abdomen, staring eyes, dull look, staggering, groaning, cessation of lactation, constipation alternating with diarrhœa, gradual wasting and prostration. Fragments of metallic lead greatly corroded by the digestive juices have been found lodged in the alimentary tract in such cases. The post-mortem appearances are not characteristic. Inflammation of the fourth stomach and intestines is sometimes, but not invariably, found. With the easily soluble lead preparations death is rapid and there is no inflammation, which, however, may be noticed after the ingestion of solid preparations.

Treatment should take the form of the removal of the cause by emetics, the pump, or saline purges. Dilute sulphuric acid or sulphates, such as Epsom or Glauber's salt, are stated to be useful by converting the lead into the form of the sparingly soluble sulphate, whilst potassium iodide is claimed to facilitate elimination.

Arsenic.—*Origin.*—Arsenic is very widely different in nature, especially as a constituent of many ores, *e.g.* of tin, lead, and zinc. As a result of this, the waters and herbage of many mining districts contain arsenic, and the principal commercial source of white arsenic (As_4O_6) is the flue-dust of the smelting furnaces. White arsenic is sparingly soluble, and the danger depends essentially on the state of subdivision. Thus it has been recorded that whereas 270 grains of white arsenic failed to kill a dog, as also did 15 grains a day over four months, $\frac{9}{10}$ of a grain of the soluble potassium arsenite killed when given by the mouth. The soluble preparations of arsenic, which are very commonly encountered in agricultural practice, are the alkali arsenites used as weed-killers and wheat dressings, and when coloured, *e.g.* by red oxide of iron, as sheep dips. Other dips, as well as alkali arsenite, sometimes contain thioarsenite, arsenious sulphide, soap, sulphur, and sometimes iron sulphate. The strength of soluble arsenic in a dip prepared for use lies between 0·25 and 0·5 per cent.

Sir Arnold Theiler (*Agricultural Journal of the Union of South Africa*, 1912, p. 321) has published the results of numerous and valuable tests to ascertain the safe doses of arsenical sulphur dip and bluestone (copper sulphate) for sheep, these being common and easily procurable remedies for intestinal parasites. Theiler concludes that 15 grains of dip is a safe dose, as also is a mixture of 15 grains of dip and 15 grains of bluestone, but a mixture of 15 grains of dip and 15 grains of bluestone to which 15 grains each of common salt and sulphur was added killed sixteen out of eighty-eight animals. The dangerous doses of powdered arsenious oxide by the mouth, as indicated above, are very variable, but Kaufmann gives the following approximations: horse, 150 to 700 grains; ox, 225 to 700 grains; sheep, 75 grains; pig, 7·5 to 15 grains; dog, 1·5 to 3 grains; fowl, 1·5 to 1·65 grains; man, 1·5 grains.

Symptoms.—The soluble arsenic compounds act rapidly, producing local irritant and remote nervous effects. In acute poisoning of the herbivoræ one notes salivation, thirst, loss of appetite, vomition when possible, loss of appetite, violent colic, fœtid diarrhœa of somewhat alliaceous odour, sometimes bloody, exhaustion, collapse, and death. One also notes paralysis of the hind extremities, coldness of the ears and horns, a subnormal temperature, trembling, stupor, and convulsions. The course of poisoning is sometimes so rapid that the animal is scarcely noticed to be ill.

A dose of 3 to 10 grains to a dog produces nausea, vomiting, moaning, hard and rapid pulse, painful evacuations, and death in convulsions in from six to thirty hours. When arsenic is absorbed through the skin a scalded appearance and sloughing in patches, especially round the eyes, over the scrotum of the bull, or udder and vulva of the cow, are observed. Chronic or slow poisoning of animals is rare. It is characterised by diminution of sensibility, difficulty of movement, and eventually entire abolition of the motor and nutritive functions. Indigestion, thirst, great wasting, and chronic disease of the joints have been observed amongst animals living near smelting works.

Intense rose-red gastro-intestinal inflammation with ecchymoses and extravasations are notable post-mortem signs of acute arsenical poisoning. There have been cases in which inflammation was not a prominent feature, but they should be viewed as exceptional. Fatty degeneration of the liver, kidneys, heart, and nervous centres is only to be expected in protracted cases. The preservation of organs heavily charged with arsenic is a notable feature.

Treatment is by emetics and purgatives, with milk, white of egg, and lime-water as demulcents. Specific antidotes are calcined magnesia and precipitated iron oxide. In an emergency precipitate tincture of iron perchloride with soda carbonate, filter through a handkerchief, and give *ad lib.* in warm water. Morphia against pain, and strychnine and ether against prostration, both hypodermically, are recommended.

Antimony. — *Origin.* — The preparations of antimony are somewhat rarely used in modern practice. Tartar emetic is the only common soluble preparation. The black sulphide, to the extent of about 18 per cent, along with nitre, sulphur, and spice, is encountered in condition powders. A strong solution of the chloride constitutes butter of antimony, used as a caustic.

The general effects of antimony recall those of arsenic, but are much less powerful. Vomition is caused by the local irritant effect, and with large doses there is also violent purging, weakness, collapse, and death. Like arsenic, antimony causes fatty degeneration of the liver and is therefore given to geese for the preparation of *foie gras*. Recently (1918) antimony has been observed in poultry foods, having probably been added with a view to improving condition and making good the poor quality of the food.

Some doubt has been expressed as to whether it is possible to kill a ruminant with antimony, but a healthy horse has been killed with 10 ounces of tartar emetic. In spite of conflicting opinions, the dangerous and often fatal effect of condition powders on horses must be taken as fully established.

Common Salt.—The question of poisoning by common salt is complicated by its confusion with brine poisoning, which is probably due to bacterial toxine action. This opinion finds support from the circumstance that the toxicity of herring brine decreases on keeping while the concentration of salt increases. It is impossible to state the dose for the pig, which has been quoted at from 4 to 8 ounces, but the writer has given six consecutive 3-ounce doses without abnormal results. Cattle have been stated to have been killed by 4-pound doses. In cattle, pigs, and fowls, salt poisoning leads to loss of appetite, excessive thirst, and frothing at the mouth. Vertigo, convulsive movements, dilatation of the pupils, blindness, and convulsions occur with the pig, similar nervous symptoms being observed in fowls. The temperature in all cases is normal or slightly depressed.

The best *treatment* of salt poisoning is abundant water. An experimental fowl ate during the course of a few hours a full poisonous dose of salt without ill effects, because it partook freely of abundant water, which was simultaneously offered.

Nitre.—The nitrates of sodium and potassium are more dangerous than common salt, a pound being poisonous to the ox, and 1 ounce to the lamb. Nitrates cause gastro-intestinal irritation, trembling, convulsions, dilatation of the pupil, and paralysis of the voluntary movements.

Sulphur.—Horses have been known to have been killed by 8 to 14 ounces each of sulphur. In the horse it causes dulness, pain, and diarrhœa, the fæces smell of sulphuretted hydrogen and are fluid, black, or grey.

In the dog violent colic, diarrhœa, and vomition, at first bloody and eventually of pure blood, with an almost imperceptible pulse and coma, were followed after twenty-four hours by recovery. Apart from intense gastro-enteritis, a smell of sulphuretted hydrogen and the possible detection of particles of sulphur are post-mortem signs.

In *treatment*, castor-oil, eggs and milk, subnitrate of bismuth, chlorodyne, and whisky have been found useful.

Phosphorus.—Ordinary or live phosphorus compounded with a suitable grease, as a rat powder, is the only phosphorus preparation likely to cause damage to animals, and then most usually with the dog, cat, and fowl.

The dangerous dose depends largely on the state of subdivision, 0·7 to 1·5 grains being given as the poisonous dose for the dog. Phosphorus is absorbed as such into the blood-stream and is given out in the exhaled breath, which therefore smells of phosphorus and glows in the dark.

Phosphorus acts as a local irritant, but as it is slowly absorbed the onset of symptoms is often delayed. Uneasiness, nausea, vomiting, and eructation ensue, with fever, thirst, and abdominal pain. In the second phase jaundice, nervous effects, delirium, convulsions, and coma precede death. The jaundice is attributed to the enlargement of the liver cells preventing the flow of bile in the ducts. Birds display great stupor or remain huddled up, beak open and comb blanched, thirst, diarrhœa, convulsions, and coma preceding death.

The prognosis of phosphorus poisoning is unfavourable.

Treatment.—The stomach should be emptied, if necessary by the tube. Copper sulphate is used both as an emetic and antidote. Old (oxidized) French turpentine is a well-known antidote of doubtful value. Avoid the use of oils which promote absorption.

Other Metals.—Some of the salts of mercury, copper, zinc, silver, barium, and chromium may give rise to poisoning. This applies to such preparations as are soluble; thus calomel and barium sulphate are harmless, unless the former is given in very large doses. Again the oxide, carbonate, and weak acid salts of zinc are apparently innocuous, the sulphate of zinc being an emetic, and the chloride a caustic irritant.

Soluble barium salts, *e.g.* the chloride, which is used as a purgative, produced poisoning resembling that of lead.

Poisoning by mercury is rare, although corrosive sublimate (the soluble bichloride) is a powerful protoplasmic poison. Mercury preparations are very rarely exposed in accessible situations, which no doubt accounts for the rarity of cases. The writer, however, once encountered a case in which the deposition of finely divided metal on herbage, as the result of an explosion in a fulminate factory, led after several months to deaths of cattle.

The soluble salts of copper, of which the sulphate, or bluestone, is a good example, are dangerous. For instance, Theiler states that 45 grains upwards causes the death of sheep from acute gastro-enteritis. The green pigment, Scheele's green or copper arsenite, is naturally very dangerous. It is not often used as a paint, except for outside work, but recent cases of damage by it have been observed.

In general it may be said that poisoning by any of the above agents pursues the course common to that noticed with the other irritant heavy metals.

Acids and Alkalis.—Brief mention will suffice of such other inorganic agents as strong mineral acids and caustic alkalis. Cases of damage by them are rare in practice, although the extension of chemical manufacturing activities in recent years leads to considerable production of contaminated effluents. Such effluents are very dilute, and calculated to produce chronic digestive trouble rather than to display the drastic corrosive effects of the strong agents. Removal of the cause is the only satisfactory counter-measure. Alkali is obviously antagonized by weak acid (vinegar), and acid by weak alkali.

Poisoning of animals by gases, such as coal gas, is rare, although poisoning by ammonia fumes has been recorded, and an extremely interesting case of the emission of fumes of hydrochloric acid from a volcanic fissure in the Kelong valley, Africa, has been described by Stordy (*Journ. Comp. Path.*, 1908, p. 75).

Organic Poisons

Under the heading Organic Poisons one includes the isolated alkaloids or extracts of medicinal plants and such artificial or natural organic products as prussic acid, carbolic acid, and the like.

Hydrocyanic Acid ; Prussic Acid

Hydrocyanic or prussic acid is one of the most powerful known poisons. It is fairly commonly encountered either from natural or artificial sources, and cyanides find extensive application in pharmacy, the electro-plating and photographic arts, and on the large scale in cyanide gold extraction.

Cyanides are produced on the manufacturing scale in the course of the deprivation of coal gas of its cyanogen compounds, and are also manufactured by other processes.

In nature hydrocyanic acid results from the fermentation by enzymes of certain glucosides, notably amygdalin of the bitter almond, the peach kernel, and other seeds; linamarin (or phaseolunatin) of the common flax, and *Phaseolus lunatus*, the Java, Rangoon bean, or Haricot de Lime, species of Vetch, and certain of the Gramineæ, *e.g.* the millet or sorghum, and maize. The various species of *Prunus* also give rise to hydrocyanic acid, and the South African *Dichapetalum cymosum*, gift-blaar or poison leaf, is very dangerous, 1½ ounces sometimes and 2 ounces always being stated to prove fatal to sheep.

The quantity of glucoside contained in the wild varieties of *Phaseolus* or Java bean, which vary in colour from pale reddish brown to purple, amounts to rather more than 0·1 per cent. It is noteworthy that under cultivation the percentage of cyanogenetic glucoside dwindles, the white haricot or butter bean being harmless. From the practical standpoint only the dark-coloured or purple beans need be regarded with suspicion. Linseed cake rarely yields more than 0·025 per cent of hydrocyanic acid, although as high a percentage as 0·055 has been noted; it is therefore only exceptionally that danger is to be apprehended from this source, as is fully borne out by practical experience in numerous feeding tests. In connection with cyanides it is well worth while pointing out that many of the numerous derivatives, in particular red and yellow prussiates, sulphocyanides, and mustard oils or organic sulphocyanides, are either comparatively harmless, or dangerous through other causes. Indeed sulpho-cyanides are constituents of human saliva though not found in that of the ox. The mustard oils are dangerous by reason of their powerful irritant effects, and do not typify cyanide poisoning. It is a great mistake often committed in medico-legal practice to class everything in which cyanide occurs as part of the name as a deadly poison. As regards toxic doses, Kaufmann gives 6 grains in the form of 2 per cent solution for the horse, and 0·6 grain for the dog.

Potassium cyanide is less toxic; thus for the dog Kaufmann gives 4·5 grains, which is equivalent to 1·75 reckoned as pure acid. A heifer examined by the writer withstood potassium cyanide equivalent to 22·5 grains pure acid, but was killed by cyanide equivalent to 30 grains taken by the mouth. Calculating on 20 grains as the minimum toxic dose for cattle, it is reckoned that two pounds of the most dangerous Java beans would be necessary to cause death, while at least eleven pounds of linseed cake would have to be used. In consequence of the rapid elimination of hydrocyanic acid, even such an unusually large feed of linseed cake would probably fail of its effect.

Symptoms.—Very large doses are rapidly fatal owing to arrest of the heart and diastole. Toxic doses, after a brief, powerfully stimulant effect, lead to depression, paralysis, and diminution of blood tension.

In cases of poisoning by cyanide-producing plants, the train of symptoms is almost always complicated by irritant effects due to other constituents, *e.g.* an essential oil in the cherry laurel. Animals poisoned by hydrocyanic acid or Java beans do not show inflammation. There is congestion of the central nervous system and the lungs; fluid, black and oily blood; the cavities of the heart contain bubbles of gas, and all parts of the corpse have a faint smell of bitter almond. As we are in possession of delicate methods in this case, it is possible to test chemically and demonstrate the presence of prussic acid even in the brain substance after minimum doses.

Treatment.—The treatment of prussic acid poisoning is exceedingly difficult. Removal of the cause by emetics or pump, and measures against prostration should be adopted. The following have been recommended: atropine; sulphide and thiosulphate of soda in the hope of producing the comparatively harmless sulphocyanides; ammonia; chlorine; anæsthesia under ether. The best-known antidote is freshly precipitated ferrous hydrate made from iron sulphate and liquor potassæ. It is given in the hope of converting the cyanide into the harmless ferrocyanide, but there is rarely time to apply the antidote in any case.

Carbolic and Tar Acids.—The pure carbolic acid is rarely used outside of the laboratory and pharmacy, and therefore is not often responsible for poisoning. The cresols (homologues of carbolic acid) form about 3 per cent of the heavy, or creosote, oil of tar. The so-called tar acids are blackish liquids running up to 25 per cent of cresols, and moreover the pure cresols are manufactured on the large scale technically.

Coal tar dips, and such disinfectants as creolin and lysol, are all of them essentially concentrated homogeneous solutions of tar acids more or less pure, with resin soaps in dips and soft soaps for higher grade preparations. They form emulsions with water.

Oil of tar, mixed with vaseline or mineral lubricating oil, to which finely divided mercury is sometimes also added, is a common mange dressing for horses. Tar acid products may also be encountered in effluent waters from gasworks, tar distilleries, and coke oven plants. The tar acids are somewhat dangerous, the Departmental Committee on tar dips having found that a 1·5 per cent tar acid dip caused grave symptoms, a 0·75 per cent tar acid dip being safe.

Symptoms.—A diluted carbolic acid after absorption exercises marked effects on the central nervous system, illustrated by weakness, stupor, and tetanic convulsions similar to those produced by strychnine, choreic movements, followed by paralysis of the locomotor system, and death.

The concentrated acid is a violent corrosive. Hobday carefully investigated the use of creolin, chinosol, and izal. With delicate breeds of dogs and cats he advises that the total application of creolin should not exceed 10 to 15 minims. In poisoning he observed subnormal temperature, paralysis of hind legs, followed by complete paralysis, prostration, and chronic spasms well marked in limbs, jaws, and eyelids. Death from collapse follows coma.

Dollar on the whole agrees with Hobday. He found that a cat of seven pounds weight, three years old, after rubbing with 5 drachms of Jeyes' in 5 per cent solution, was poisoned and died within fourteen hours. He argues, however, that this preparation is no worse than any other lotion or efficacious agent.

Treatment.—In treatment sodium sulphate is given in the hope of promoting the formation of sulpho-carbolates, which are easily eliminated. An emetic of zinc sulphate is recommended for small animals, followed by egg albumen in milk and a mixture of chlorodyne and lime water. With large animals strong purgatives and whisky have proved successful when the purge acted. Oil of turpentine has been usefully employed.

Strychnine.—*Origin.*—This alkaloid occurs, together with brucine and igasurine, as the active principle of the seeds of varieties of *strychnos*, particularly *strychnos nux vomica* of East Indian habit. The powdered seed constitutes nux vomica powder. The amount of strychnine varies from 0·5 to 2 per cent. Brucine is present in larger proportions, has the same general actions, but is less powerful.

Strychnine is a very common constituent of vermin powders, in which it is mixed with starch or flour, and blue or soot. The existence of these preparations renders strychnine an accessible poison, and makes it responsible for a large proportion of cases of poisoning among dogs, cats, and foxes. It is also used to protect stacks from vermin, and as a grain dressing. In this latter respect advantage is taken of the fact that birds are relatively immune, and that whereas sparrows may be destroyed, fowls may escape.

Strychnine is very toxic, and the poisonous doses are fixed by Kaufmann as follows: horse, 3 to 4·5 grains; ox, 3 to 6 grains; pig, 0·15 to 0·7 grain; dog, 0·075 to 0·3 grain. For the dog this is roughly between one-thirteenth and one-third of a grain. Putting the dog at 0·75 the equivalent dose for man is 0·4, and for the fowl 2 grains, which well shows the relative order of sensitiveness. In practice strychnine is only given to dogs in small and carefully regulated doses, one-hundredth of a grain being given with caution to toy varieties. One-thirtieth of a grain has caused strychnine spasms in a fox terrier.

Symptoms.—Strychnine is a powerful stimulant to the central motor cells, and greatly increases the reflex irritability. After paralysis of the peripheral nerve-endings by cocaine, injection of strychnine fails to produce the tetanic convulsions. Under the influence of strychnine such a gentle stimulus as a slight air current induces a normal reflex immediately followed by the characteristic general tetanic spasms, during which the back is curved, respiration is arrested, and the muscles are tense. Death results from asphyxiation, the respiration usually ceasing after two or three spasms. During the spasms the rigidity of the extended limbs is so great that a small animal may be lifted in a perfectly straight position by one extremity. Macqueen observed strychnine symptoms in the treatment of paralysis in the horse by doses increasing from 1 to 5 grains twice daily. Twitching of the superficial muscles is a preliminary warning. The horse rears, falls and makes galloping movements, so that it moves backwards on its side in a circle. Tobacco gives relief. Dogs become very uneasy, whine, are nauseated, and sometimes vomit. The general tetanic spasms occur with increasing violence at intervals of one, two, or more minutes until death, which is rapid after the first onset of symptoms.

Post-mortem reveals the appearances of asphyxia, *rigor mortis* is characteristic, but its absence is not a decisively negative sign. The feet, or in birds, the claws, are generally incurved, and the muscles of the jaws are rigid.

Treatment.—Unless the dose is large, and provided the cause is promptly recognized, treatment is hopeful. Remove the cause, giving a tenth to half a grain according to size, of apomorphine as an emetic. Chloroform inhalation, or morphine, is useful, but chloral, 6 to 20 grains, according to size, by the mouth, is the best physiological antidote. In giving antidotes by the mouth there is danger, for if a spasm occurs the liquid may pass into the trachea and suffocate. Remembering that morphine causes excitation in the lower animals, the use of chloral is greatly to be preferred.

Morphine and Opium.—*Origin.*—Morphine forms the chief constituent of the alkaloids of the Opium poppy, *Papaver somniferum*, native to Southern Europe and the Levant. Codeine and narceine are also hypnotics, whilst thebaine, papaverine, and narcotine are convulsants.

The lower animals, by reason of their inferior cerebral development, withstand far larger doses of morphine than man. The doses ordinarily given to dogs for anæsthetic purposes would prove fatal to a man. Four grains per pound body-weight represents a fatal dose for the dog. To kill the horse 75 to 100 grains would be needed. Sixty grains given by the mouth to a pony evoked no marked symptoms, and, after slaughter, impaction of the stomach contents due to gastric paralysis was the only sign of action. The symptoms of nausea, indigestion, and tympany are due to the arrest of the digestive functions. The excitant effects of morphine are conspicuous in the lower animals. In the ox and horse the animal moves in a circular direction, butting regularly into obstacles, and giving the general impression of madness or marked delirium. This phase may be protracted, and is eventually followed by coma, respiratory failure, and death. The excitant effects of morphine are enhanced in the action of the allied heroin (diacetyl morphine). In *treatment* remove the cause, combat excitement by cold applications to the head and depressant effects by caffeine. The stimulation of small doses of atropine seems useful.

There is very little chance of the accidental poisoning of animals by morphine or the allied alkaloids, and the same holds for such alkaloids as cocaine, eserine or physostigmine, pilocarpine, emetine, gelsemine, veratrine, curarine, etc., particularly as none of the plants from which these alkaloids are obtained is indigenous to the temperate zones. They are a part of pharmaceutical equipment, and as such are, or should be, under control.

Poisonous Plants

Anything approaching a full enumeration of the plants which are definitely known as poisonous, still more those to which suspicion of causing damage attaches, would be altogether outside the scope of an article like the present. Particularly in view of the existence of such a comprehensive work as that of Cornévin, *Des plantes vénéneuses*, which covers European and many North African species, and having regard further to the somewhat monotonous uniformity in the recorded symptoms alleged to result from the ingestion of different plants, a list in this place of the principal suspicious species and varieties is amply sufficient. On the other hand, however, there are certain genera, regarding whose toxicity long experience is fortified by concrete illustrations in point, and more detailed reference will therefore be confined to them, alluding in the first instance to British and North European plants.

Coniferæ.—Members of the Pine family are the source of turpentine, and the Coniferæ in general are distinguished by containing essential oils such as savin from *Juniperus sabina.* The poisonous effects of overdosage of turpentine have long been recognized, large doses causing intestinal irritation and sometimes ulceration. After absorption the oil exercises paralysant effects in the same order as those of alcohol. *Oil of savin* or savin tops enjoy repute as abortives. Horses sometimes fall the victims of poisoning through eating cuttings. The general effects are those of an irritant, and are treated by means of opium, aperients, demulcents, and stimulants.

The order Coniferæ includes yew, or *Taxus baccata*, and varieties, which are common trees, and which, apart from containing irritant oils, contain the very toxic non-irritant alkaloid taxine. A remarkable feature of yew poisoning is its rapidity; often, whilst quietly chewing, cattle drop as if shot, and have been known to die while in the act of eating the plant. After eating yew, an animal will stop suddenly, start blowing and trembling, fall, and die quietly within five minutes, with symptoms like apoplexy. The alkaloid paralyses the alimentary system with stoppage of digestion and consequent symptoms. Intense inflammation of the stomach is almost invariably observed, but it rarely extends to the intestines. The prognosis is very grave.

Treatment.—A soda bicarbonate emulsion of one pint of linseed oil with two ounces each of chlorodyne and nitrous ether, followed by whisky and linseed oil, and later stimulants and chlorodyne, has been successful. If the diagnosis is certain, Wallis Hoare recommends rumenotomy.

Ranunculaceæ.—This order comprises the medicinally very important aconite. *Aconitum napellus*, Monk's-hood or Wolf's-bane, is a plant of cultivation in Great Britain. The root contains the exceedingly poisonous alkaloid aconitine, which acts as a gastro-intestinal irritant, and exercises its general effects upon the medullary vagus centre, producing cardiac depression and fall of blood pressure, and on the respiration, the breathing becoming slow. Poisoning by aconite has been observed in the horse, and is marked by champing and copious salivation, eructation of frothy matter and continued attempts to vomit. The gait is staggering, and the pulse weak. Paralysis follows, with difficult respiration and weak heart and pulse, dilated pupils, blanched membranes and low temperature. There is loss of power and sensation, convulsions, and death by asphyxia.

Apart from *general treatment* the depressant action may be combated by means of digitalis, ether, or atropine.

This order also includes the black and green Hellebores, which should not be confused with the white Hellebore, *Veratrum album*, belonging to the Colchicaceæ.

Hellebore acts as an irritant and at the same time on the heart in a somewhat similar manner to digitalis.

Members of the genus *Ranunculus*, which are common weeds of cultivation, contain acrid juices whose chemical nature is not well ascertained. All the species are to be regarded as suspicious. *Ranunculus* first produces gastro-enteritis, colic, nausea, vomiting if possible, salivation, emission of black fæces, and sometimes hæmaturia. There are nervous symptoms, with retardation of pulse, slow and stertorous respiration, weakness of the posterior parts, difficulty in mastication and drinking, and blindness.

Treatment.—J. Gerrard successfully overcame common buttercup poisoning of the horse by nitrous ether, aromatic ammonia, extract of hyoscyamus, peppermint water, tincture of opium, and a four-drachm ball. In spite of the extreme commonness of the varieties of Ranunculus it is not freely taken by animals, except in case of scarcity or when they are confronted by the plant for the first time.

Cruciferæ.—The Cruciferæ include mustard. White and black mustard flower each contains a glucoside giving rise on fermentation to the respective essential oils of mustard, oxybenzyl sulpho-cyanide from the white, and allyl sulpho-cyanide from the black. It has long been known that the whole seed is quite harmless, passing through the system unaltered. Whether the dangerous effects of crushed mustard seed are due to the essential oils or to some other constituent, it remains a fact well established as far back as 1861 that the presence of crushed mustard seeds in oil cakes, notably rape, makes them exceedingly dangerous. About a pound of such cake may cause fatal poisoning. J. Gerrard in 1875 gives the following picture of the poisoning of cattle: uneasiness, restlessness and intense colic, with frantic rushing about and mania, ending in exhaustion, falling, struggles, and collapse. Gerrard quotes this as a good example of an irritant poison. His narrative is almost textually reproduced in very recent accounts received from different sources relating to mustard cake poisoning. Even a press cake of mustard husks after milling has produced similar effects. In all such cases the rumen contents smell of oil of mustard, and complaint is sometimes made by the men engaged in handling the corpses, of the stinging effect on the thin skin of the fore-arm. The frantic mania, which is characteristic of mustard poisoning, was not noticed in a case of illness among cattle eating wild mustard on pasture. The cause in the latter instance was probably different.

Leguminosæ.—Amongst the Leguminosæ reference has been made, under "Hydrocyanic Acid," to poisoning by the Java or Rangoon bean.

The yellow lupin is held responsible for sheep poisoning on the Continent, and according to the German authorities a daily ration consisting of a pound of the whole plant, three-fifths of a pound of empty pods, or half a pound of the seeds, will produce poisoning, marked by inappetence, dyspnœa and fever, hæmaturia, circulatory and digestive derangements, trembling spasms, and vertigo. Jaundice is characteristic.

Lathyrism.—Several varieties of the genus *Lathyrus*, such as Dogtooth, Indian or Mutter pea, are dangerous. Their ill repute indeed goes back to the days of Hippocrates and Pliny. The cases of poisoning amongst horses appear to have been first recorded in England in 1886. The causation of lathyrism is obscure, no certain conclusions as to the active principle having yet been reached. Lathyrism is only produced when considerable portions of the pea enter into the rations over a prolonged period of time—in man about four months, in the horse fed exclusively on the pea, ten days, but when one to two quarts only are given daily, towards the eightieth day. The malady may declare itself as long as fifty days after the cessation of the feeding.

Paralysis of the lower extremities is a constant sign of lathyrism in man. M'Call in 1886 placed on record complete observations on the horse. An animal was feeding well but thick in its wind. After going about two hundred yards with an empty lorry it stood with the fore-legs forward, neck stretched, elbows out, and laboured breathing. It was with difficulty kept on its feet until unyoked, and each breath gave a loud sound from the larynx. There was profuse sweating, quick, irregular and intermittent pulse, increased impulse of the heart, venous pulsations, normal temperature, and a vibration over the region of the larynx. The symptoms disappeared after about five minutes, and the animal began to eat hay. Later accounts substantially agree. Sometimes the paroxysms prove fatal, otherwise there is a speedy temporary recovery. Change of diet, at any rate at first, does not arrest the malady.

With sheep and pigs and experimental dogs, paralysis of the hind extremities is notable.

The only effective treatment, besides removal of the cause and rest, appears to be tracheotomy.

Loco disease is common in Colorado and Montana, and as with lathyrism the etiology is obscure, but no doubt is felt that the disease is caused by leguminous plants of the Astragalus species.

In acute locoism in sheep the animal becomes unmanageable, completely blind and dizzy, walking in long circles to the right, and then standing for a few moments in a stupor. The head is elevated and drawn to the right at the beginning of an attack, and these become more frequent as the malady progresses. The expression and pulse are nearly normal, and the pupil is not dilated. Trembling fits are characteristic and the animals tend to bolt in an erratic manner.

Java bean poisoning.—See "Hydrocyanic Acid," *ante*.

Umbelliferæ.—*Conium maculatum*, or Spotted Hemlock, is the source of the volatile alkaloid conine, and as a common weed has caused dangerous poisoning of the larger animals. The general effect of conine is that of a paralysant. In the horse the plant produces nausea, accelerated respiration and dyspnœa, muscular trembling beginning in the posterior members, and paralysis. There is loss of sensibility, subnormal temperature, rapid pulse, and death by arrest of respiration. In cattle, profound stupor and arrest of digestion; sometimes constipation, but in other instances bloody evacuations.

Treatment is by evacuation of the stomach and purgation; tannic acid to remove the alkaloid; warmth and stimulants.

The same natural order includes *Cicuta virosa*, Cowbane or Water Hemlock, whose root is very dangerous. The active principle, cicutoxin, is a powerful narcotic. Cobbold observed a case in Brittany where eleven beasts died with violent symptoms of vertigo. Animals appear to die without a struggle.

Œnanthe crocata, or Water Dropwort, has in its root œnanthotoxin, apparently closely resembling cicutoxin in its effects. In 1835 a gang of convicts at Woolwich suffered from poisoning from this cause. *Œnanthe* appears also to possess irritant principles. Wallis Hoare observed well-marked delirium followed by rapid death in cows poisoned by roots left in a field after a flood. In the horse nervous symptoms predominate, and with the pig large doses kill with the rapidity of cyanide.

Ericaceæ.—This family includes the ornamental shrubs, Rhododendron, Azalea, and Calmia. Rhododendron leaves are dangerous. In cattle and sheep they produce intense pain, diarrhœa and discomfort, gritting of the teeth, salivation, and frequently vomition. There is cessation of lactation, trembling, spasms, loss of power, and death. Brisk oleaginous purgatives, followed by chlorodyne or counter-irritants to abdominal pain, followed by stimulants and tonics, prove successful in the treatment of Rhododendron poisoning.

Solanaceæ.—The four British genera *Atropa*, *Hyoscyamus*, *Datura*, and *Solanum* comprise some of our commonest poisonous plants. To them may be added the exotic tobacco plant, *Nicotiana tabacum*. These may be subdivided according to the active principles into the Atropine group—*Atropa belladonna*, *Hyoscyamus*, and *Datura*, which contain the mydriatic alkaloids atropine and its congeners; the Solanine group—*Solanum dulcamara*, or Bittersweet, and the potato, containing the glucosidal alkaloid solanine; tobacco, containing the volatile alkaloid nicotine analogous to conine.

The most dangerous of these is *Atropa belladonna*, or Deadly Nightshade, which frequents waste and stony places in chalky districts. By reason of its scarcity, and further because most of the atropine is in the roots, cases of poisoning by the plant are very rare. The possibilities of poisoning by the pharmaceutical preparations atropine and extract of belladonna are naturally more restricted. Atropine inhibits secretion and leads in animals to dryness of the mouth, and causes an increase in temperature and pulse and respiration. There is dilatation of the pupil, blindness, nervousness, delirium, and muscular trembling. After the period of excitement there is fall of temperature, convulsions, motor and sensory paralysis, feeble and slow respiration, relaxation of the sphincters, and death in convulsions. One grain of atropine sulphate subcutaneously evokes symptoms in the horse. The pig, goat, sheep, and rabbit are said to be tolerant to the root, but the alkaloid on injection causes typical symptoms. Belladonna liniment sometimes produces symptoms in the small animals either by reason of absorption or more usually of licking.

Hyoscyamus niger, or Black Henbane, containing principally the alkaloids hyoscyamine and hyoscine, acts as more powerfully depressant than *Atropa*. In the very rare cases of poisoning by it, in contradistinction to poisoning by atropine, profuse salivation has been observed.

Datura stramonium, or Thorn-apple, is rather rare in England, but is a common weed in the United States known as Jimson weed, and also in South Africa, where it is called Stinkblaar. Poisoning by it resembles that due to atropine, which is in harmony with the fact that the active principle, formerly regarded as a single substance and called *Daturine*, is a mixture of the mydriatic alkaloids atropine, hyoscyamine, and hyoscine. The post-mortem appearances of poisoning by these plants are not characteristic, being those of asphyxia.

In *treatment*, in addition to removal of the cause, sedatives may be indicated in the early stages of excitement; in the later stages stupor is combated by movement and stimulants. Eserine is the physiological antidote.

Cases of poisoning by *Solanum dulcamara* and the potato are probably due to the same cause. The unripe and green potato, as well as the old, rotten, or sprouting tuber is dangerous.

In poisoning by the potato, all accounts agree as regards the general narcotic effect. In general there is depression, loss of appetite, cessation of lactation, gritting of the teeth, and profound prostration with remarkable somnolence. Mydriasis is not always observed. A period of constipation is generally succeeded by diarrhœa, and, when possible, vomition. In less acute cases, to the dominant feature of prostration there are added intestinal irritation and rapid loss of flesh.

The post-mortem appearances are not characteristic.

In *treatment* strychnine subcutaneously is indicated and has proved successful. In the few recorded cases of poisoning by bittersweet, greenish diarrhœa is in addition noticed.

Cases of *tobacco poisoning* have been observed, and usually result from the use of tobacco juice as an external application. The main features in such cases are profuse sweating, tremors, nausea, disturbance of the respiration, and dilatation of the nostrils; later there is profound prostration and deep coma. Strychnine has been found useful in the treatment of nicotine poisoning in the dog.

Scrophularineæ.—The commonest poisonous member of this family is *Digitalis purpurea*, or Foxglove, whose leaves and seeds are the principal sources of digitalis. The toxic doses of the green leaves are small. Cornévin quotes four to five ounces for the horses, six to seven for the ox, one for the sheep, and a half to three-quarters for the pig. Digitalis is an irritant, but its important physiological effect is on the heart, causing increased and prolonged systole with diminished and shortened diastole. In poisoning, the heart is arrested in systole. It is a powerful diuretic, tends to be cumulative, and is contra-indicated in kidney disease. Hard breathing, erratic feeding, lassitude, an almost imperceptible pulse, contracted pupil, and excessive urination have been noticed in cattle and horses after eating hay containing dried foxglove. In horses which had eaten foxglove there was observed sleepiness, swollen eyelids, dilated pupils, normal respiration, pulse 65 to 75, and the second heart sound frequently obliterated; later the breathing became laboured, head immensely swollen, tongue greatly enlarged and protruding, pulse 80 and most erratic, great restlessness. The respiration became more difficult and stertorous, tongue and buccal membranes livid, jugular, standing out.

In a case of the poisoning of pigs by decoction of *digitalis* leaves the chief feature was languor and sleepiness. The animals refused to eat or drink, attempted to vomit, and repeatedly passed small quantities of fæces. Curiously, urination was scanty and strained.

Treatment consists of the administration of tannic acid followed by an oleaginous purge and general stimulants.

Euphorbiaceæ.—The most important dangerous plants of this family are the exotics *Ricinus communis* or castor, *Croton tiglium* or croton, and *Jatropha curcas*, or purging nut.

Many species of Euphorbia, *e.g.* *Euphorbia hibernica*, or Irish Spurge, which is used for poisoning fish, contain acrid juices and may be dangerous. The South African *Euphorbia genistoides* or Piss grass contains an acrid irritant juice. The outstanding feature of its action is severe urethritis, the attempts to urinate being frequent and painful. The animal lies down, the bladder becoming more distended, and dies in coma, or in a violent effort at relief.

Castor Poisoning.—The castor-oil seed is extensively imported for the sake of the valuable oil used not only in pharmacy, but also as a lubricant for high-speed machinery. The press cakes are unfortunately useless as food-stuffs, since they contain the exceedingly dangerous toxine, ricine. It should be noted that the ricine is contained in the body of the seed, and not in the husk. As indicated in the introduction to this article, this and other vegetable toxines are bodies of unknown constitution but extraordinary physiological activity, analogous to snake venoms, and they serve in a manner to bridge over the gap between the relatively simple poison and the bacterial toxine. Like the latter they are distinguished by giving rise on sublethal dosage to specific antibodies, and by being destroyed by heat, 56° C. being the minimum lethal temperature. Unfortunately, however, especially when dealing with a mass in the dry state, it is exceedingly difficult to ensure complete destruction of the ricine, even at a steam heat, although this has been successfully done on a large scale. An animal immunized to ricine is able to withstand four to eight hundred times the ordinary full toxic dose. Its serum then contains a specific antibody which is able to protect a second subject against ricine only, but not against crotine or any other toxine, and which gives a precipitin reaction with ricine extract. As is to be expected from a toxine poison, enormously greater doses are required by the mouth than on injection.

Castor seeds have produced fatalities among horses and cattle. In horses the poisoning is marked by extreme dulness, loss of appetite, coldness, abdominal pain, and constipation. Purgation has only been noticed in relatively few instances, and greatly assists recovery. With cattle, diarrhœa and bloody purgation have been held to be characteristic, but this is not in accordance with the facts. On experimental tests we have noticed pale and fœtid diarrhœa in young calves after one ounce of whole seeds. Pressed seeds up to eighteen ounces tended to make the fæces dark or hard. The ordinary small seed is less toxic than some of the larger varieties (*R. sanguineus—R. Zanzibariensis*) which are cultivated for ornamental purposes. Fourteen and sixteen ounces respectively of these seeds killed where eighteen ounces of the ordinary seeds had failed. Diarrhœa was not marked, nor were the evacuations bloody; the general toxic action preponderates over the local irritation. In croton poisoning the effects of the drastic irritant croton oil predominate.

Treatment. — Morphine sulphate, or the equivalent of tincture of opium in drenches of mucilage, cornflour, or arrowroot, have been recommended successfully in treatment.

Many plants and foods have been reputed to be poisonous, amongst them cotton cake, brewer's grains, and beet pulp residues. It seems doubtful in all these cases whether there is any definite poisonous principle at work.

Undecorticated cotton cake may cause impaction.

Beet residues and beet molasses are very rich in saline constituents; in order that they may be utilized as food-stuffs it is necessary to bring the animals on to them by cautiously feeding small quantities in the first instance.

Acorn Disease.—The remarkable disorder known as acorn poisoning affects cattle, but not sheep, deer, or swine. Its features were first clearly investigated by Simmonds and Brown in 1871. The symptoms are not declared until after the acorns have been digested and expelled. In the early stages there is constipation, succeeded by persistent diarrhœa, with frequent small dark and bloody evacuations. There is loss of appetite, suspension of rumination, wasting, colic, and excessive urination. The temperature may be subnormal. Soreness of the mouth, pallor of the membranes, and a discharge from the nose and eyes, which are sunken, are noticed. The lesions are in general those of an irritant poison. *Treatment* is by oleaginous purgatives and opiates.

Bracken.—Bracken poisoning is another obscure malady. Storrar in 1893 drew attention to the poisoning of cattle by bracken, and was inclined to attribute it to the indigestibility and not necessarily to any specific toxic effect. The symptoms summarized in the 1909 Report of the Chief Veterinary Officer of the Board of Agriculture are: Temperature, 104° to 107° F., loss of appetite, blood-tinged discharge from the mouth and nose, blood from the bowels, pallor of the membranes of the eye: great depression, coma; and death in from twelve to seventy-two hours after the onset of symptoms. The disease produces congestion of the stomach and intestines, and notably there are hæmorrhages in the heart and body muscle and under the skin. It is suggested tentatively by the Board that the weed tormentil, *Potentilla tormentilla*, which occurs along with bracken in many localities, may be in part responsible.

G. D. L.

A List of Common Poisons and their Antidotes

Poison.	*Antidotes.*
Acids	Weak alkalis, bicarbonate of soda, chalk or whitening; milk and eggs.
Alkalis	Weak acids, vinegar; oils.
Aconite	Digitalis; alcohol; ether.
Arsenic	Precipitated iron oxide; emetic or oil; white of egg.
Barium	Glauber salts; Epsom salts; weak sulphuric acid.
Belladonna and Hyoscyamus	Eserine; caffeine; stimulants; oils; forced exercise.
Carbolic acid and Creolins	Glauber salts; turpentine; saccharated limewater; egg and milk; stimulants.
Castor seeds	Morphine; oils.
Common salt	Plentiful supply of water; oils.
Digitalis (foxglove)	Tannic acid; oils; stimulants.
Hemlock	Tannic acid; purgative; stimulants.
Lead	Glauber salts; Epsom salts; dilute sulphuric acid; potassium iodide in chronic cases.
Mercury; copper; and other metallic salts	Epsom salts; white of egg and milk.
Morphine and opium	Atropine; caffeine; forced exercise.
Phosphorus	Copper sulphate; old turpentine; *avoid* oils and ether.
Prussic acid and Java beans	Ferrous hydrate; hyposulphite of soda; atropine.
Strychnine and Nux vomica	Chloral hydrate; bromides; chloroform inhalations; tannin.
Sulphur	Bismuth subnitrate; chlorodyne; alcohol (brandy or whisky).
Tobacco	Strychnine; caffeine; brandy.
Yew	Chlorodyne; bicarbonate of soda in oil; rumenotomy in cattle.

G. H. W.

FOOD AND THE PRINCIPLES OF FEEDING

Food may be defined to be any solid or fluid taken into the body for the purpose of growth and repair of tissues and the production of heat and energy. This definition excludes the atmosphere. There has been some controversy as to whether the atmosphere ought not to be included as food, because undoubtedly the body receives from the atmosphere an amount of oxygen which is useful both for growth and repair and the production of heat and energy, but it is generally agreed that food only includes what passes into the gastro-intestinal tract. The health and usefulness of the lower animals are to a great extent a question of proper feeding. The principles of feeding are the same with all the lower animals, although this article will specially deal with horses.

There is no doubt that considerable economy could be effected in most large stables were the principles of feeding properly understood and applied. It should first of all be grasped that the body makes the best use of food if the different food constituents are in certain proportion to each other; that if they are not, some portion of the food is either wasted or may even produce disease. It should also be remembered that the horses or cows in the stable have no power of selection. They have to eat what is given them or go without.

The different constituents of food are familiarly known as the *proximate principles*, some of them complex chemical compounds but elementary so far as the building up of the body is concerned. Every blade of grass and every seed of corn contain these proximate principles but in varying amounts, some are rich in one and poor in another, others contain them in the correct proportions. These proximate principles are first of all divided into two classes, the *organic* and the *inorganic*, the inorganic comprising the mineral salts, to which reference will be made more fully later.

The organic proximate principles are divided firstly into those containing nitrogen and those without nitrogen. Those containing nitrogen are known as **proteids** or **albuminates** or often albuminoids. Chemically they contain the elements carbon, hydrogen, oxygen, nitrogen, sulphur, and occasionally phosphorus. The chemical composition of a proteid is $N_{16}C_{54}O_{22}H_7S_1$. Proteids are divisible into two great groups according to their nutritive value. The more nutritious consist of albumin, glutin, casein, myosin, and legumin. The other group, less nutritious, is more concerned with the food of carnivora and consists of gelatin (from boiling bones and cartilage), chondrin, ossein, and keratin. Glue is really an impure form of gelatin got from boiling horns and hoofs.

Albumin is the essential constituent of that substance which, called by physiologists protoplasm, really is the physical basis of life. The white of egg is almost pure albumin. Glutin is the name given to the albumin of most cereals, and can easily be demonstrated by placing some wheat flour in a muslin bag and running a stream of water through it. The milky water passing from the bag is due to the presence of starch, the sticky substance left behind in the bag is nearly pure glutin. Myosin is the name given to muscle or flesh albumin, casein to the albumin of milk. It is the curd which is formed when an acid is added to milk. Legumin is the albumin of the pea and bean tribe (Leguminosæ). Avenin is the albumin of oats and gliadin that of wheat. Most of these albumins are coagulable by heat. Casein is an exception.

The passage of a food proteid through the body is as follows: It is unacted upon in the mouth. In the stomach, by the action of the gastric juice containing pepsin and hydrochloric acid, it is converted into what is known as peptone, which is a soluble form of proteid, and passes from the stomach into the bowels, whence it is absorbed by the lacteals and passes into the blood. Such of the proteids that are not digested by the gastric juice are further subjected to the digestive action of pancreatic secretion containing a ferment "trypsin," and by intestinal secretion (succus entericus). The functions of albumin in the body are especially to build up the muscular tissue, to determine the absorption of oxygen, provide heat and energy; and if the starches, sugars, and fats are deficient in the food, it can be converted into fat. It is not completely oxidized in the body, but is excreted by the kidneys in the form of urea, which on exposure to air, as is very evident in most stables, is rapidly converted into carbonate of ammonia. If given in excess it does not seem to be stored up in the body for future use in the way that the fats are, but rather tends to produce disease. The proteid is the only organic compound in the food containing nitrogen.

The second of the proximate principles consists of the **fats** and **hydrocarbons.** They consist of carbon, oxygen, and hydrogen in the proportion of $C_{10}H_{18}O$. In true hydrocarbons there is no oxygen. The fats are compounds of glycerine with the fatty acids (oleic, margaric, and palmitic). Their functions are to repair and renew fatty tissues, and they yield energy and heat. They cannot be converted, so far as is known, into proteids as they do not contain the necessary chemical elements. Their passage through the body is as follows. They are unacted upon in the mouth or the stomach. On leaving the stomach for the small intestine they are immediately met by the flow of bile from the liver, and with the help of the bile they form an emulsion which is capable of absorption by the lacteals, and so reach the blood. Fat is also acted upon and rendered absorbable by steapsin contained in pancreatic secretion. As has been mentioned, they give heat and energy, repair and renew the fatty tissues, and are completely oxidized into carbonic acid (CO_2) and water, in which forms they are excreted from the body. If given in excess they are stored up for future use, especially in the form of glycogen in the liver and as fat throughout the greater portion of the body. An excess of fatty food does not produce anything worse than a want of condition.

The third of the proximate principles is the **carbohydrates** represented by the starches and sugars. These consist of the same chemical elements as the fats, namely, carbon, oxygen, and hydrogen, but in this case the hydrogen and oxygen are always in the proportion to form water (H_2O). Thus starch has the chemical composition $C_6H_{10}O_5$, cane sugar $C_{12}H_{22}O_{11}$, and grape sugar $C_6H_{12}O_6$.

The carbohydrates constitute the bulk of nearly all natural foods. In their passage through the body the starches are acted upon in the mouth by ptyalin, a ferment contained in the saliva, and are more or less, according to the length of time passed in the mouth and the amount of saliva secreted, converted into glucose or dextrose, otherwise known as grape sugar. The sugars are merely more or less dissolved by the saliva. There is no action on either the starches or sugars in the stomach. On reaching the small bowel they meet with the secretion of the pancreas, whose starch-digesting ferment "amylopsin" completes the conversion of the starches into sugar. This on passing into the blood is carried along to the liver, where it is retained as glycogen, to be supplied to the needs of the body as required. The functions of the carbohydrates are to supply heat and energy. They are unable to form proteids, but they can be converted into fat, indeed it is probable that the most of the body fat is derived from the carbohydrates. Like the fats they are completely oxidized in the system and leave the body in the form of carbon dioxide and water.

The fourth proximate principle consists of **cellulose** and **woody fibre** or lignin, which form the framework of plants. They are true carbohydrates and have the same chemical composition as sugar, but are classed separately because they are supposed to be of no nutritive value. Not that there is no digestion or solution of cellulose, but because the energy required to digest it is equal to the energy derived from it after digestion. The general opinion, however, amongst veterinarians is that in cattle at any rate there is a nutritive value in cellulose. Amongst the constituents consisting of carbon, oxygen, and hydrogen should also be mentioned the various **vegetable acids,** which are very useful in stimulating digestion.

The *inorganic portion* of the food consists of the **mineral salts,** of which perhaps common salt or sodium chloride is the most important to the adult animal. It not only assists in keeping in solution the globulins of the blood, but it is the source of the hydrochloric acid in the gastric juice which materially aids the conversion of the insoluble proteid in the stomach into the soluble peptone.

The complete withholding of common salt from food will cause the death of the animal from starvation, owing to the proteid matter not being dissolved. Common salt does not exist in sufficient quantity in the natural foods of herbivora for hard-working horses or heavily milking cows. To these animals, therefore, an addition should be made to the food of common salt or in the form of rock salt in the manger. Phosphates, calcium, potassium, sodium, magnesium, and iron salts are all essential to the repair and growth of different parts of the body. The phosphates of lime, potash, and magnesium go to the formation of bone and also nervous tissue. Muscular tissue is also rich in potash. Iron helps to form the hæmoglobin of the blood.

"The last proximate principle is **water,** and when we remember that almost two-thirds of the body is composed of water there is no difficulty in comprehending its necessity as a food. Its use in the body is for the solution and conveyance of the food to different parts of the system, the excretion of effete products, the equalization of heat by evaporation both from lungs and skin, as well as the regulation of all the chemical and mechanical functions of the body" (Notter and Firth).

A complete food, therefore, must consist of proteids, fats, carbohydrates, salts and water. No two cereals contain these constituents in the same amount or proportion. Thus, peas and beans contain a high percentage of proteid, linseed contains a high percentage of fat, while maize contains a high percentage of soluble carbohydrate, so one has next to consider the "nutritive relation" or the proportion in which the proximate principles of food are arranged towards each other, and what are the best ratios or proportions of the albuminoids (proteids), carbohydrates, fats, and cellulose for a food to supply its highest economic value, while at the same time sustaining the animal in perfect health.

In the case of carriage horses and hunters it is more a matter of health and condition without regard to cost, but in the case of large horse owners such as municipalities, large carriers or railway companies, it is a question of how to get the work done by the cheapest method of feeding consistent with the good health of the horses. I am acquainted with veterinary surgeons who in the first year of their appointment have saved for their employers more than the whole of their salary, not only without the horses suffering in the least, but with an actual improvement in health as shown by the number of working days. Let us then try to understand what is meant by the **nutritive relation** or **ratios** of food, which seems to be something of a "pons asinorum" to the beginner, though it is really very simple.

There are three ratios to be considered: the first known as the **nitrogenous ratio,** the second the **fatty ratio,** and the third the **complete nutritive ratio.**

The **nitrogenous ratio** means the ratio or proportion of digestible albuminoids to the digestible non-albuminoids, cellulose and salts not being included. In other words, it is the proportion of the proteid to the fats and carbohydrates. Perhaps the following table or classification of the constituents of food will enable this to be more easily understood.

- Organic
  - Nitrogenous or Proteids
  - Non-Nitrogenous
    - Fats or Hydrocarbons.
    - Carbohydrates (starches and sugars).
    - Cellulose.
    - Water.
    - Vegetable Acids.
- Inorganic: Mineral Salts.

The nitrogenous ratio then would be represented as follows:

$$\frac{\text{Proteid}}{\text{Fats} + \text{Carbohydrates}}.$$

In order to arrive at this, it is necessary to know the percentage composition of the food, which is ascertained by taking the mean of a large number of chemical analyses. For instance, take the analysis of oats. The percentage composition of oats is as follows:

Water . .	13·7
Nitrogenous Matter	12·0
Fats . .	6·0
Carbohydrates .	56·6
Cellulose . .	9·0
Salts . . .	2·7
	100

The formula will stand thus:

$$\frac{12}{6+56\cdot 6}=\frac{12}{62\cdot 6}=\frac{1}{5\cdot 2}$$

This means that for every one part of nitrogenous matter there are 5·2 parts of non-nitrogenous matter. By this calculation, therefore, the nitrogenous ratio of oats would be said to be 1 : 5·2. This is not quite correct, owing to the fact that the nutritive value of fats is nearly $2\frac{1}{2}$ times the value of carbohydrates, and it is necessary in order to be correct to reduce these two to the same value. What has to be done, therefore, is to multiply the percentage of fats by $2\frac{1}{2}$. The correct formula will stand thus:

$$\frac{\text{Proteids}}{(\text{Fats}\times 2\frac{1}{2})+\text{Carbohydrates}}=\frac{12}{(6\times 2\frac{1}{2})+56\cdot 6}$$

$$=\frac{12}{15+56\cdot 6}=\frac{12}{71\cdot 6}=\frac{1}{5\cdot 96},$$

or almost as 1 to 6.

The **fatty ratio** is simpler still. It is the proportion of fats to nitrogenous matter only, and is represented by the formula $\frac{\text{Proteids}}{\text{Fats}}$. Looking again at our percentage composition of oats we find it consists of 6 per cent of fats and 12 per cent of nitrogenous matter, the fatty ratio will be as follows: $\frac{6}{12}=\frac{1}{2}$ or 1 : 2. In this case of course the fats become the numerator instead of the proteid. This means that for every two parts of nitrogenous matter in oats there is one part of fat. The fatty ratio is therefore said to be 1 : 2.

The **complete nutritive ratio** is arrived at by ascertaining the proportion of nitrogenous matter to the fats and carbohydrates plus the cellulose. Thus

$$\frac{12}{(6\times 2\frac{1}{2})+56\cdot 6+9}=\frac{12}{15+56\cdot 6+9}=\frac{12}{80\cdot 6}=\frac{1}{6\cdot 7}.$$

The complete nutritive ratio of oats therefore would be 1 : 6·7. This third or complete nutritive ratio is not considered of great importance in human hygiene, but it is very important to the lower animals, as the bulk of the food is increased by the cellulose, and bulk is necessary for good digestion in the herbivorous animals.

Let us now work out the ratios of a sample of hay having the following percentage composition:

Water	14·3
Proteid . .	8·2
Fats . . .	2·0
Carbohydrates . .	41·3
Cellulose . . .	30·0
Salts .	4·2

The nitrogenous ratio works out as follows:

$$\frac{8\cdot 2}{(2\times 2\frac{1}{2})+41\cdot 3}=\frac{8\cdot 2}{5+41\cdot 3}=\frac{8\cdot 2}{46\cdot 3}=1:5\cdot 6.$$

The fatty ratio is $\frac{2}{8\cdot 2}=1:4\cdot 1$.

The complete nutritive ratio is

$$\frac{8\cdot 2}{(2\times 2\frac{1}{2})+41\cdot 3+30}=\frac{8\cdot 2}{76\cdot 3}=1:9\cdot 3.$$

Let us now take a mixed diet and work that out.

The Army ration in peace time is 10 lbs. of oats and 12 lbs. of hay.

	Oats.	Hay.
Water	13·7	14·3
Proteid	12·0	8·2
Fats	6·0	2·0
Carbohydrates	56·6	41·3
Cellulose	9·0	30·0
Salts	2·7	4·2

We shall now have to multiply the percentages by the number of lbs. of each material.

The following is the nitrogenous ratio:

$$\frac{120+98{\cdot}4}{(60\times 2\tfrac{1}{2})+(24\times 2\tfrac{1}{2})+566+495{\cdot}6}$$

$$=\frac{218{\cdot}4}{150+60+566+495{\cdot}6}=\frac{218{\cdot}4}{1271{\cdot}6}=1:5{\cdot}8.$$

The following is the fatty ratio:

$$\frac{60+24}{120+98{\cdot}4}=\frac{84}{218{\cdot}4}=1:2{\cdot}6.$$

The complete nutritive ratio, which is the nitrogenous ratio plus the cellulose added to the denominator, is

$$\frac{218{\cdot}4}{1271{\cdot}6+90+360}=\frac{218{\cdot}4}{1721{\cdot}6}=1:7{\cdot}8.$$

These ratios are all ideal for adult horses. That is to say, the nearer the nitrogenous ratio works out to as 1 : 5½, the better it is. The fatty ratio is not so important and may work out anywhere between as 1 : 2 and 1 : 3. The complete nutritive ratio should work out something between 1 : 6·5 and 1 : 8 for horses and not less than 1 : 8 for cattle, though the ration just considered does not take cattle into consideration. Now provided this is as cheap a ration as you can get, there is no better or healthier food for horses.

Substitutional Dieting.—There are generally various cereals on the market which by substituting for the whole or a portion of the oats will give a quite healthy mixture and capable in the case of draught horses of giving the necessary amount of work, and it is the business of the food expert to discover the cheapest food capable of fulfilling the required conditions. This will vary according to the markets. Sometimes oats are relatively higher in price than maize or barley. Sometimes a few beans may with advantage form a part of a horse's ration, but beans being highly nitrogenous would unbalance the food unless given in small quantity or some substitute found containing less nitrogen. It is a rather interesting study to watch the markets and try and work out a well-balanced food at the lowest possible cost. By a *well-balanced food* is meant a food in which the proximate principles are in their proper proportion, *i.e.* where the ratios work out correctly. I have worked out a diet (see below) as an example which usually is not an expensive one comparatively, and which I have found a singularly healthy one. With the necessary care in feeding and watering to which I shall refer later, it has almost abolished colic, enteritis, and that most unsatisfactory mischief known as twisted colon, which I am convinced is due in most cases to errors of feeding, more

RATIOS[1] OF A DIET CONSISTING OF 6 LBS. OF OATS, 6 LBS. OF MAIZE, 3 LBS. OF BEANS, 2 LBS. OF BRAN, AND 13 LBS. OF HAY

	Nitrogenous.	Fats.	Carbo-hydrates.	Cellulose.	Percentage digested.		
					Albuminoids.	Fats.	Starches.
6 lbs. of oats	12	6	56·6	9	87	78	76
6 lbs. of maize	10	6	61	2	77	63	94
3 lbs. of beans	25	2	50	3	86	8	93
2 lbs. of bran	14	4	50	10	75	50	70
13 lbs. of hay	8·2	2	41·3	30	60	20	52

Nitrogenous ratio:

$$\frac{72+60+75+28+106{\cdot}6}{2\tfrac{1}{2}\,(36+36+6+8+26)+(339{\cdot}6+366+150+100+536{\cdot}9)}=\frac{341{\cdot}6}{280+1492{\cdot}5}=\frac{341{\cdot}6}{1772{\cdot}5}=\frac{1}{5{\cdot}18}\text{ or }1:5{\cdot}18.$$

Fatty ratio:

$$\frac{36+36+6+8+26}{72+60+75+28+106{\cdot}6}=\frac{112}{341{\cdot}6}=\frac{1}{3}\text{ or }1:3.$$

Complete nutritive ratio obtained by adding cellulose to fats and carbohydrates:

$$\frac{341{\cdot}6}{1772{\cdot}5+54+12+9+20+390}=\frac{341{\cdot}6}{2257{\cdot}5}=\frac{1}{6{\cdot}6}\text{ or }1:6{\cdot}6.$$

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[1] Not corrected for digestibility, see page 524.

particularly with regard to the balance of the food. It consists of the following mixture: 6 lbs. of oats, 6 lbs. of maize, 3 lbs. of beans, 2 lbs. of bran, and 13 lbs. of hay.

In the summer time, when less food is required to keep up the body temperature, a portion or even the whole of the beans may be omitted.

In this food for cart-horses I advocate 10 lbs. of hay given as chaff amongst the corn ration and 3 lbs. of long hay in the rack at night for the following reasons. A horse is almost compelled to grind this food, he cannot bolt it. Many horses prefer corn to hay, provided both are of good quality, and will eat the whole of the corn before touching the hay and then perhaps not eat enough hay to balance the corn, or if horses are not allowed a sufficient time for their meal they have not time to eat the hay after finishing the corn. (The man who comes late in the morning is often responsible for this.) But by mixing it in this way the horse has always a fairly balanced ration, and the 3 lbs. of hay at night in the rack will give him something to play with during the night if of a wakeful disposition and prevent him eating his bedding. Working cart-horses should be allowed at least two hours for breakfast before commencing work and not less than one and a half hours for the mid-day meal. They should be fed four times a day, the last feed as late as convenient at night. Ten pounds weight of food is too much to put into a horse's small stomach at once. That is what three meals a day means, and is the cause of a good deal of stomach and bowel trouble. Food may be damped to prevent a horse blowing it out of the manger. It should never be made very wet except in the case of a bran or linseed mash. Very wet food is a temptation to the horse to bolt his food. Grinding the food is necessary to excite salivation, without which the starches are not acted on in the mouth. Boiled food is only permissible to old horses with defective teeth or some such special reason, as when colts have tender gums from teething. There are two useful rules to remember, in working out a mixed chop for horses, which may save a good deal of calculation and worry. Never include in a suggested food more than 3 lbs. of peas or beans per diem or there is difficulty in balancing the food, and bowel affections are the result; and never let your hay fall below the proportion of as three is to four of corn in weight for the same reason.

It will have been noticed that at the end of the table giving a suggested mixed chop for heavy horses I have also shown the percentage digested of each of the proximate principles. If the absolutely correct ratios are desired, the percentage digested must be taken into consideration, but it complicates the calculation and in my opinion, at any rate, is unnecessary. I have tried a number of mixed foods and worked them out according to the percentage digested, and found the difference was only a matter of one or two decimals, which is of no importance in the composition of a diet.

The Watering of Horses.—Horses should always be watered before being fed. A considerable quantity of fluid is required for the production of gastric juice, and watering before feeding helps this process. Watering after feeding not only dilutes the gastric juice and so weakens its action, but will also wash through the stomach a quantity of undigested food, the nutritive value of which is thus lost even if nothing worse in the form of bowel trouble occurs. There is some prejudice against giving a horse water when he is sweating. It is an overrated prejudice, but where such exists it is surely not a difficult matter to take the chill off the water, which abolishes the objection.

Digestibility of Foods.—Every natural food contains some matter which is absolutely indigestible; but more than this, every food has a portion of that which is digestible unacted upon, and which is excreted from the system without undergoing absorption. Our knowledge of the digestibility of food by farm animals is almost entirely derived from German investigators, and for those who would wish to refer further to the subject I would recommend the translation of the German work of Professor Emil Wolf by H. M. Cousins, M.A., entitled *Farm Foods.*

The general method of investigation has been to supply an animal with weighed quantities of food, the composition of which has been ascertained by chemical analysis. During this experimental diet the solid excrements are collected and weighed, and finally analysed by the same chemical methods previously applied to the food. Subject to certain small corrections for intestinal secretions, we obtain by this plan the amount of each constituent of the food which has passed through the animal unabsorbed, and, by difference, the amount digested.

The amount of food digested seems not to differ whether the animal is in an emaciated state, requiring all the nourishment possible, or whether it is in a condition of being overfed. If a piece of meat is placed in a large amount of gastric juice no more of it is digested than if placed in what is only just a sufficiency.

The proportion of each constituent digested for 100 parts supplied is known as the *digestive coefficient.* The mean coefficient of digestibility of proteids in the horse is 69, of fats 59, and of carbohydrates 68. This means that of every hundred parts of each of these substances the horse can digest $\frac{69}{100}$ of the first, $\frac{59}{100}$ of the second, and $\frac{68}{100}$ of the third. The ox digests a greater percentage than the horse. Hard work with exhaustion seems to diminish the coefficient of digestion.

In hay the digestibility varies according to the ripeness of the grasses when cut. The young plant is always more digestible than the mature one, and in overgot or overripe ryegrass hay for example the amount of soluble carbohydrates has been so greatly transformed into cellulose as to render it a most indigestible food. Boiling the food does not increase the coefficient of digestion in the least but rather decreases it.

It is also generally accepted, though not universally, that in a mixed food the digestibility of each food is increased by the admixture of the other. That is to say, supposing a horse is thrown off work for some time and only hay and water given, which is sufficient for a subsistence diet, it would actually be cheaper to add a couple of pounds of oats per day, because by so doing the animal would extract so much greater nourishment from the hay. Further experiments, however, are required on this subject before one can be dogmatic upon it.

Heat and Energy Production.—I have now to say a few words on the heat- and energy-producing power of foods. This has been calculated especially by the researches of the late Dr. Joule of Manchester. It is a chemical law that the complete combustion of a certain amount of material of any description, whether coal in a fire or food in the body, always gives off a definite amount of heat; whether the combustion be slow or quick the same amount of heat is given off in the aggregate. Joule proved that heat can be converted into energy without loss, and he found from an elaborate series of experiments that the amount of heat required to raise 1 lb. weight of water through one degree Fahrenheit, *i.e.* to raise the temperature from say 39° F. to 40° F. for example would, if converted into energy, be sufficient to lift 772 lbs. weight one foot in height.

The amount of heat necessary to raise 1 lb. of water one degree higher in temperature is known as the heat unit or calorie, and the mechanical equivalent of one degree of heat in the Fahrenheit scale is said to be 772 foot pounds.

Joule's experiments prove that 1 oz. of albumin, of which only $\frac{2}{3}$ is oxidized in the body, yields nearly 170 foot tons of potential energy.

One ounce of fat oxidized in the system yields 378 foot tons of potential energy.
One ounce of starch oxidized in the system yields 138 foot tons of potential energy.
One ounce of grape sugar oxidized in the system yields 124 foot tons of potential energy.

These calculations, so far as the lower animals are concerned, are of more academic interest than practical importance; so much energy is required in keeping up the body temperature and in the movements of the heart, lungs, bowels, and general animal metabolism that it is calculated that only about $\frac{1}{7}$ of the food consumed is available for work. For example, the horse requires food sufficient to produce 21,000 foot tons of energy for the processes of the body alone, and if you calculate that a horse's day's work should approximate to 3000 foot tons, you must then give him an amount of food sufficient to produce 24,000 foot tons of energy.

It will be noticed in the above statements that the term "potential energy" is used. Perhaps a few words in explanation may be useful. Energy means the power of producing movement, or so far as we are concerned the power of producing work. It is divided into two kinds, potential and kinetic. Potential energy is energy which is stored up for future use and is not being expended in work at the moment. One example of potential energy usually given is poising a stone above the head; release the stone and the potential energy becomes converted into kinetic energy or actual movement. This is, I believe, one of Newton's own examples, but I must confess it always seems to me a better example of gravity or the attraction of bodies to each other. Another example is, if you wind up a watch spring, you can do it so carefully that when wound it will remain still, but touch the watch spring with a needle and it will commence to work, and thus the potential energy becomes converted into kinetic energy; so, fat may be stored up in the body and give to that body potential energy, which, if you work the animal on an insufficient supply of food, will be used up by the body to produce the kinetic energy or *vis viva*.

The kinetic energy of an animal is represented by (1) his maintenance work, *i.e.* the work expended in breathing, circulation, and the production of animal heat, and (2) by the external work he does, as moving the limbs, drawing a load, and carrying himself. This energy is produced by the breaking down of the protoplasm of the cells and the combustion of food materials.

Various Articles used as Food

Grass

Grass, the natural food of herbivora, is made up of a large number of different species of Graminaceæ, and included amongst the grasses but usually referred to as "artificial grasses" are the clover, vetches, sainfoin, and lucerne, belonging to the natural order Leguminosæ. The grasses vary considerably in their feeding value and are divided into three groups, namely: "good," "indifferent," and "bad or useless."

Roughly speaking, the composition of grass is as follows:

Water	80 per cent.
Proteid	3 to 5 per cent.
Fats	1 per cent.
Carbohydrates	10 to 12 per cent.
Cellulose	3 to 12 per cent.
Salts	2 per cent.

Grass itself is quite sufficient to keep a horse in health provided that no hard work is required, but the quantity of water produces a condition known as hydræmia or excess of water in the blood, a condition quite healthy but not favourable for work. It will be noticed that when a horse comes up from grass, unless in a very dry season, he will not touch water for two or three days, and it is for this reason that a watery *purge* is valuable before putting him to work. Otherwise "filled legs," if nothing worse, is apt to supervene.

Classification of Grasses

- Good
  - Meadow Foxtail.
  - Timothy or Cat's-tail.
  - Crested Dog's-tail.
  - Fescues.
  - Rye Grasses.
  - Artificial Grasses.
- Moderate
  - Cocksfoot.
  - Sweet Scented Vernal.
  - Yellow Oat Grass.
  - Fiorin.
- Useless
  - Poas.
  - Yorkshire Fog.
  - Hair Grass.
  - False Oat Grass.
  - Quaker Grass.
  - Couch Grass.

Meadow Foxtail (*Alopecurus pratensis*).—One of the earliest grasses, flowering in April, and one of the best, especially for pastures. If sown in meadows it has usually lost its seed before the time for mowing, but even then it loses less of its nutriment than most of the other grasses. Either nitrate of soda or sulphate of ammonia has a very beneficial effect on its growth. Meadow foxtail is three years before attaining its full growth.

Timothy or Meadow Cat's-tail (*Phleum pratense*).—A hardy good grass but late, flowering in July; attains its greatest height and productiveness in the first year after being sown. All good meadow hay should have a fair proportion of timothy. This grass might be mistaken for foxtail, but is easily distinguishable by the absence of the hairy awns which are quite a feature of the foxtail.

Crested Dog's-tail (*Cynosurus cristatus*).—A good grass, which does not, however, add great weight to the hay. It has a rather small flowering head compared with the two previous grasses, but is good both for pastures and hay. It responds better to nitrate of soda than to sulphate of ammonia.

Fescues.—The Fescues are all classed as good grasses with the exception, perhaps, of the Sheep's Fescue (*Festuca ovina*), which is so short that it is useless for hay, as it is too short to be mown either with a machine or a scythe.

Rye Grasses (*Lolium italicum* and *Lolium perenne*).—The Italian ryegrass is a biennial, *i.e.* it only lasts for two years, and should not be sown in pastures. It is a good grass, but requires to be cut early. In Scotland it is grown as a separate crop and is estimated very highly. In other parts of the country, as a rule, it is sown along with red clover and is commonly known as first crop. There is no better hay than clover and ryegrass, assuming, of course, that it is well got. Both this grass and the red clover flower a second time in the same season and so produce two crops of hay, hence the names first and second crop. The *Lolium perenne* or perennial ryegrass is not perennial on sandy or very light soils, but otherwise is correctly named, and is one of the very best of grasses. In some parts grown as a separate crop, it is generally to be seen and should be present in all good meadow hay. The two grasses can easily be distinguished from each other by the presence of the hairs or awns on the Italian variety which are absent in the perennial.

The Artificial Grasses.—Clover, lucerne, vetches, and sainfoin belong to the natural order Leguminosæ. They are all good. Some of the clovers are perennial, but the red clover grown with ryegrass is at most biennial, as it is incapable of self-fertilization, depending for this process on the bee carrying the pollen to the female flowers. There is, however, a perennial red clover known as cow grass or zigzag clover, grown principally in the south of England, where it seems to be highly thought of. The white or Dutch clover and the Alsike are both perennial. Neither horses nor cattle seem to be specially fond of these varieties, although their nutritive value is quite good. Lucerne, a very ancient plant described by writers long before the Christian era, is a valuable forage plant, but is not much grown in this country, as it requires a dry climate and a dry soil. It is usually eaten green in this country. It is an excellent forage grass and produces three or four cuttings in the year. It is imported into this country as hay from the South American states under the name of Alfalfa.

Vetches, a good food when young, but indigestible when they commence to go yellow at the roots, are eaten green and usually sown with an admixture of oats round the headlands of cornfields.

Sainfoin is also a valuable forage plant, largely grown in the south of England, and also eaten green or made into hay.

Cocksfoot (*Dactylis glomerata*).—This grass has a very wide distribution, is very hardy, and usually classed as a good grass. This view, however, requires some qualification. It is a good grass if grown alone and cut early, but usually it is sown with the late meadow grasses, and being an early flowering grass it has not only lost its seeds, but in the stems the digestible carbohydrates to a remarkable extent become converted into cellulose and even lignin, an advanced stage of cellulose which is absolutely indigestible. From a farmer's point of view also it is not too desirable, as its roots spread in the form of a cushion and interfere with the full growth of the adjoining grasses.

Sweet Scented Vernal (*Anthoxanthum odoratum*).—This is the grass which gives the pleasant smell to meadow hay. It has no smell when green but only when dried. It is a moderate grass from a nutritive point of view, and as it flowers early it has frequently lost its seeds before being harvested. The essential oil is supposed to act as a condiment, and in some parts of Europe is extracted for the manufacture of scent.

Yellow Oat Grass (*Avena flavescens*).—I am inclined to rate this grass somewhat higher than most authorities, and I have no objection to quite a fair quantity of it in a sample of hay.

Fiorin or Creeping Bent Grass (*Agrostis alba*) comes rather low down amongst the moderate grasses.

Poas.—There is a considerable number of varieties of Poa all more or less resembling each other. They are only regarded as very moderate grasses, although personally I prefer them to fiorin.

Yorkshire Fog or Woolly Soft Grass (*Holcus lanatus*).—This is a grass which one sees in far too great quantity in most samples of meadow hay. It is a useless grass, although in Australia it was for a time rather highly thought of, but I believe that now they are coming to the same conclusion as authorities in this country.

The other grasses in the list classed as useless need not be further alluded to. They are all found more or less in inferior samples of meadow hay.

To all interested who may wish to recognize these and other grasses at sight I would strongly recommend the purchase from Messrs. Sutton, of Reading, or some other seed-growers of a box containing samples of the different grasses, which can be had for a few shillings. It is very much easier to learn them from these samples, which are labelled, than from any book plates.

Hay

Hay is usually divided into three classes, Upland, Meadow, and Water Meadow, but generally speaking, I think, a better division is that commercially adopted, *i.e.* the division into First Crop and Meadow Hay.

The first crop consists almost entirely of Italian ryegrass and red clover. It usually follows an oat, wheat, or barley crop and is very good, especially for milch cattle. It has not been considered suitable for racehorses or hunters, owing to its larger proportion of cellulose than in hay containing the permanent grasses. At one time no stud groom would have used this hay for galloping horses, but this prejudice is not so pronounced as it used to be, and now in many racing and hunting stables it is freely used. This ryegrass and clover hay produces a good aftermath or second crop, as both the ryegrass and the clover flower a second time. In the case of the permanent grasses many of them do not flower a second time, with the result that the aftermath of meadow hay consists almost entirely of the stems and leaves of the grasses, and the absence of the flowering heads detracts very seriously from the nutritive value of the hay.

How to make good Hay.—In grass cut too young, *i.e.* before it flowers, much of the nourishment is in the roots, and the nitrogenous portion existing in the stem consists only of amides. It has not yet become a true proteid, and amides can only be converted into heat and energy. The difficulty of saving such hay, also, would be very great and the shrinkage excessive owing to the large amount of water it contains. Young grass cut at the proper time, *i.e.* after flowering but before the pollen begins to fall, contains a great deal more nutriment than in the case of grass cut late. When the grass has done growing the soluble carbohydrates (starches and sugars) in the stem become converted into cellulose to build up and strengthen the structure of the stem; cellulose is indigestible fibre, and the indigestible fibre of young hay is therefore much less than in hay cut late. Old grasses have well-developed seeds, and the nutriment, especially the proteid matter which was originally in the stem, will have passed into the seeds, and when the seeds fall this most valuable portion of the food is lost altogether. By letting the flowering heads ripen and fall, a considerable amount of the nitrogenous matter extracted from the stalks will be lost. Of course, the barometer has always something to say as to the exact day when one can commence cutting hay, but there is considerable difference in the feeding value of hay cut from old grass. Many farmers, particularly those who do not consume their own hay, allow it to get too ripe, because they get a greater weight per acre, and the general public is too ignorant to appreciate the difference in value. Hay made from old grass also often contains more weeds, such as docks and sorrel. Often in the market one can see two loads of hay selling at about the same price per ton where the nutritive value of the one is 20 or 30 per cent more than the other. Hay as made in England should never be eaten until it has been at least three months in the stack. It takes nearly this time before the temperature of the centre of a stack is reduced to that of the surrounding atmosphere, owing to fermentative changes, and if used before is a prolific cause of digestive trouble.

Good hay should have a rather greenish tint, hard long stems, clean, fresh, and possessing the well-known aroma. Meadow hay should have a considerable variety of good grasses and an abundance of flowering heads.

When hay is stacked a bacterial fermentation takes place, producing heat and a change of a portion of the starchy material into sugar. If there is too much moisture left in the stems when stacked, *i.e.* if stacked before it is ready, this fermentation is carried a stage further, the hay becomes dark in colour and the sugars are converted into acetic acid, or the stack may even take fire from spontaneous combustion. Horses will often for a while eat readily of this dark-coloured or *mow-burnt hay* if the process has not gone too far, but good buyers will reject it, as it is a prolific cause of diuresis (profuse staling) and excessive thirst, followed by want of condition and emaciation.

Mouldy hay is an entirely different condition from mow-burnt hay and is due to a fungus (*Penicillium glaucum*) which finds a congenial habitat on hay which has received a shower of rain during the process of housing or stacking.

That hay which is the cheapest to make is always the best, by which I mean the hay which has required the least turning over, the least labour. Hay that has been more than three or four days between cutting and housing and exposed to bad weather has lost a considerable amount of its soluble carbohydrates and loses its crispness. It is usually darker in colour, does not rustle when handled, but feels soft and woolly. On the other hand, hay left too long in the sun becomes bleached, brittle, and dusty, and loses its flavour and quality.

The Grains

The grains are really not the natural food of herbivora, but are necessary when work is required. Hay of itself will only give little over a subsistence diet, *i.e.* the amount of nourishment necessary to subsist without any extra exertion.

Oats.—Of the grains the most valuable and widely used is undoubtedly oats. There are numerous varieties of oats, the commonest being the white, yellow polands, tawny, black tartary, and crosses between the white and black oats such as the tartar kings and storm kings. The white oats and the crosses are the commonest all over England and Scotland, while the black oats are mostly grown in Ireland. There is very little to choose between equally good samples. The true test is the amount of oatmeal which a sample will yield, but generally speaking a good sample should be large in the grain, short, plump, thin in the skin or husk, bright in colour, and feel hard like small pebbles when handled. They should weigh not less than 40 lbs. to the bushel. Oats vary somewhat in their chemical composition, but the following may be taken as the analysis of an average sample :

Water	13·7
Proteids	12
Fat	6
Carbohydrates	56·6
Cellulose	9
Salts	2·7

Oats are divided into new and old. They are classed as new until they are a year old. It is unnecessary for oats to be a year old before being used, but it is a very good rule never to allow them to be eaten in the year they are grown. As soon as January sets in they may be eaten with impunity.

There has been some controversy as to the economy of crushing oats. The late Professor Pritchard contended that it was unnecessary. He held the view that the horse had a splendid grinding machine in his mouth, and that even in case of a horse bolting his oats, the heat and moisture of the stomach were sufficient to burst the grain and enable the animal to extract all the nourishment. This is not correct. I tested it by using the dung of a horse that habitually bolted his oats as a manure for a flower garden, and grew quite a considerable quantity of oats amongst the flowers. But while I contend it is an economy to crush oats I never advocate the purchasing of crushed oats from a dealer, for the following reasons. A dealer will naturally not crush his best oats, a sample of light oats is not so easy to distinguish when crushed, and in the case of a dishonest dealer such as unfortunately does occasionally exist, there may even be added to the sample the husks left in the process of preparing oatmeal. It is a somewhat informing study to examine the proportion of empty oat husks in many samples of crushed oats bought from dealers. Of course I do not say this is general, but I do say it occurs sufficiently often to be worth looking into.

New oats are occasionally thrashed before they have been long in the stack, and are then *kiln dried* and rushed on to the market before the price comes down. They can be distinguished by being a little puckered in the husk from the heat, from being softer than the old, and from having, mingled with the earthy smell of new oats, a slight "sulphury" odour. Old oats as a rule from attrition and dryness have lost their beards or tips. This should not be confounded with what is known as *clipped oats*, in which the ends have been taken off by machinery, leaving them quite square at the pointed end. This process is adapted particularly with Canadian and New Zealand oats, which are imported into this country in large quantities. They are very light oats, often weighing not more than 35 lbs. to the bushel. They are generally well got, and with the exception of being badly fed, a good oat, but should always be fed by weight or the animal will go short of his supposed ration. When oats have suffered from over-heating in the stack, a condition comparable to mowburnt hay, they become, except in the case of black oats, darker in colour, and are termed "foxy" from the characteristic smell. These will produce the same effects in horses as mowburnt hay. In order to hide this condition they are often bleached by means of sulphur dioxide (doctored oats). This can be detected by rubbing a small sample between the palms of the hands, when the smell of the SO_2 is easily discerned.

Good oats and hay, with an occasional bran and linseed mash, are a sufficient food to keep in health and condition all but those horses doing exceptionally hard work, and other grains are only substituted on the ground of economy.

Maize or Indian Corn.—Maize is often called Indian corn and is largely grown in India, but the principal portion reaching this country comes either from Russia or America. The *Russian grain* is smaller and rounder compared with the American flat corn, and is mainly used for feeding poultry. It is the principal substitute for oats, but is unsuitable for horses doing fast work, as although it has a fair proportion of proteid, it is exceptionally rich in carbohydrates and is consequently rather fat-forming than muscle-producing. It is exceptionally poor in salts and is practically without lime. It should therefore not be used in great quantity to infoal mares, or to young growing stock requiring lime for the formation of bone. It will be remembered by those who had much experience of American horses, that many went lame if put to fast work for the first few months after their arrival in this country, from a form of ostitis or inflammation of the bones of the fore-legs, particularly due, in the opinion of many, to the softness of the bone from deficiency of lime salts, due to having been reared largely on maize. After a few months in this country, and being fed on oats, this tendency to lameness disappeared. For cart-horses maize may be substituted for oats to the extent of one-half the oat ration, but should always be crushed, or much of the hard grain will pass through the body without digestion. In South Africa maize is known as "mealies" and is extensively used as a horse food. As a food for cattle in the form of maize-meal it is exceedingly useful both for milk and fattening purposes, and it is a capital food for fattening pigs, though practically for the last fortnight of a pig's life oatmeal should be given, when the flavour of the bacon is greatly improved.

The percentage composition of maize is as follows:

Water . . .	12·7
Proteids .	10·1
Fats	4·7
Carbohydrates . .	68·6
Cellulose . .	2·3
Salts . . .	1·6

Barley.—When the market conditions are favourable barley may be substituted for oats. Mr. J. Malcolm, F.R.C.V.S., of Birmingham, who had exceptional experience in feeding cart-horses, stated that he could find no difference between two stables in one of which barley had been substituted entirely for oats. My experience has not quite coincided with his. I have thought that after a few weeks' substitution it produced a certain amount of bowel trouble, due, as I considered, to the awns on the barley. Barley should be crushed, otherwise with greedy feeders a proportion of the hard grain will pass through the body without digestion. It is an excellent food in the form of meal for either cattle, sheep, or pigs, and in the case of young colts two to four years old during the eruption of the molar teeth and consequent tenderness of the gums. It is quite easily assimilated when boiled. This is one of the few conditions when boiling of grain is permissible and indeed advisable.

The following is the analysis of barley:

Water . . .	14·3
Proteids . .	10
Fats	2·3
Carbohydrates .	64·1
Cellulose .	7·1
Salts .	2·2

Wheat.—Wheat is an unfit food for horses, under any conditions, unless eaten green or baked into bread. It can produce tympany, laminitis, and stomach staggers. It is fortunate that its demand for human food is such as to preclude the probability of its use as a horse food. Damaged wheat is sometimes used for cattle, but with them, unless in very small quantity, is apt to produce impaction of the rumen. The percentage composition will show at once how difficult it is to balance a food if wheat forms one of the component parts, but it is the gluten of the wheat which forms a gummy mass in the stomach and especially is the cause of trouble.

Its analysis is as follows:

Water	14
Proteids	14
Fats	1·5
Carbohydrates	67
Cellulose	1·5
Salts	2

Bran is the outer covering of the kernel of wheat, and is known as red or white bran, according to the colour of the wheat from which it is obtained. Commercially there are two kinds of bran, broad and fine. The broad bran with large flakes is the best, and lends itself least to adulteration. The best bran has a small amount of the flour adherent to it, but these small particles do not tend to form glutinous masses in the stomach as in the case of a feed of wheat, although large quantities of dry bran are not advisable. Two pounds of dry bran mixed with the other food is quite sufficient for a day's ration for either a horse or cow. Large quantities of dry bran have been blamed for the formation of calculi in the bowel, owing, it is said, to the quantity of salts, especially phosphates, which it contains. If, in addition to dry bran, the water in the district is very hard from the presence of lime salts, I am of opinion that the two together do constitute a danger.

There has been considerable controversy as to the value of bran as a food. F. Smith in his *Veterinary Hygiene* says it is useless, whereas experiments at the West of Scotland Agricultural Society point to its being highly valuable. It is a very healthy food for sick animals in the form of a mash made with boiling water and then allowed to cool slowly, and has a somewhat laxative effect due, according to some, to the irritating effect of the bran on the bowels, but according to later opinion, due to the magnesium constituent of the bran (Phytin). It is also a good food for young animals from its digestibility and richness in phosphates, but it should be remembered in this connection that the phosphate is that of magnesium, and that bran is poor in lime salts. Its analysis is as follows:

Water	13·90
Proteids	14·90
Fats	3·60
Carbohydrates	51
Cellulose	10·49
Salts	5·70

Bran lends itself to adulteration principally from the addition of sand or sawdust or both. By stirring up some bran and water the sand can be detected by immediately falling to the bottom, whereas sawdust will float after the bran has settled down. Good bran should have a sweet and pleasant smell. Sometimes one notices an oily smell, in which case it has probably been mixed with some bran which has been used for cleaning machinery. This probably would do a horse no harm if he would eat it, but he wont.

Beans.—Horse beans (*Faba vulgaris*) are largely grown in the British Isles, and are also imported in large quantities from abroad, especially the East. They belong to the natural order Leguminosæ, all of which order are very rich in proteids, and generally speaking, with the remarkable exception of Soya beans, very poor in fats. Three lbs. weight per day is the maximum quantity permissible for either horses or cattle. They may be given whole or split to horses mixed with other food, and in the form of meal to cattle. English beans are much to be preferred to foreign. Foreign beans are often a distinct danger from the presence of small pebbles amongst them, due to the method of threshing the beans in the open on the sandy soil, and then shaking in a large meshed sieve or riddle which gets rid of most of the sand but leaves the small stones. These are a distinct danger from two reasons, firstly, horses' teeth are often split from trying to grind the small stones, and secondly, from their liability to form a nucleus for calculi formation in the large bowels. Foreign beans are also often affected by the weevil, a small insect, quite visible in size. Indeed, some weevils reach up to ⅛ of an inch in length. They bore into the bean, and may destroy the whole of the inside, leaving nothing but the shell. Their presence is recognized by the existence of a small hole about the size of a pinhead. They are extremely difficult to get rid of. You cannot drown them. Beans affected with weevil may be left under water all night and the weevil is still alive. Indeed the only method of dealing with weevily beans is to grind the bean into meal.

The following is the percentage composition of beans:

Water	14·4
Proteids	25·0
Fats	1·6
Carbohydrates[1]	55·8
Salts	3·2

Peas.—The variety used for feeding the larger animals is principally the white pea, *Pisum arvense.* Peas are not largely grown in the British Isles, but are principally imported from Canada and the East, especially India. The

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[1] In this analysis, the cellulose has apparently been included with the carbohydrates. The average amount of cellulose is about 7 per cent, which would leave in the above sample 48·8 per cent of digestible carbohydrate.—G. H. W.

Canadian peas are large and in every way much the best of the foreign peas. Indian peas are smaller, and can be recognized by the presence, in almost every sample, of a number of small brown peas (*Pisum sativum*). Nearly every sample of Indian peas contains a large quantity of sand, and they are an unsafe food for horses unless first dealt with by a powerful dust extractor. I have known as much as 25 per cent of Indian peas to consist of sand. In other words, for every four sacks of Indian peas you are probably buying one of sand.

Peas are not so good as beans for horses, being less easily digested and productive of tympany. For cattle, in the form of meal, they may be used up to 2 lbs. weight per day. Peas, like beans, are also very subject to attacks of weevil.

The following is the percentage composition:

Water	14·4
Proteids	22·6
Fats	1·9
Carbohydrates	53·0
Cellulose	5·4
Salts	2·7

Soya Beans — Manchurian Beans. — These beans, ivory coloured and looking like ovoid peas, have a most extraordinary percentage composition, containing as much as 40 per cent of proteid and 25 per cent of fat. They are largely imported from Japan and Manchuria for the purpose of extracting the oil for soap-making, after which they are pressed into a cake. They have also been crushed and sold as Soya bean meal or cake. It is possible that experiments will show that if properly balanced by the admixture of other foods, a valuable feeding stuff may result, but until this is done they can only be considered as a very dangerous food, and one that should only be given in the smallest quantity, certainly not more than 1 lb. weight per day.

Gram (*Cicer arietinum*).—Gram, a grain of the leguminous order, is grown and largely used in India. It is a brown wrinkled kind of pea, and its analysis is very similar to beans. It has been for some years largely imported into this country, and is used crushed as a substitute for beans and in the same quantity. Provided it is only used in limited quantity, it seems to have no ill effects, but to be quite a good food.

Rye.—Rye is only grown in certain parts of the British Isles, and is not largely used as a food. It is very subject to the attack of a fungus known as Ergot (*Claviceps purpurea*), and when affected with this should never be used to parturient animals, as the action of ergot of rye upon the uterus is apt to produce abortion.

Linseed.—Linseed is the seed of the flax plant, and is a valuable food for both sick and healthy animals. It should never be given dry, as in this case many grains will pass through the body without solution. It may be given mixed with bran or other food either boiled, scalded, or stewed. Stewing is the best method of dealing with linseed, as here the grains are not burst so much as in boiling, and are consequently more palatable to the animal. It forms the bulk of most of the calf meals, and in the case of an orphan foal oil-dust gruel, *i.e.* gruel from ground linseed from which the bulk of the oil has been expressed, can be used as a substitute for milk, though not entirely, owing to its laxative properties.

The following is the analysis of linseed:

Water	12·3
Proteids	20·5
Fats	37·0
Carbohydrates	19·6
Cellulose	7·2
Salts	3·4

Hand-Feeding a Foal. — Here, perhaps, I might interpolate that the common method of feeding an orphan foal is to take cow's milk, add boiling water until the temperature of the mixture is 100° F., and a teaspoonful of sugar to the pint of milk. In this connection it should be remembered that cane sugar should not be used; lactose, of course, is ideal, but its expense is prohibitive. Brewers' sugar (glucose) acts very well, and is easily assimilated by the young animal.

Poisonous Grain, etc.

There are poisonous grains which only require a few words, as they are sufficiently understood by grain merchants in this country to prevent their importation in large quantity unless as an adulteration to some other ground food or meals.

Java Beans.—These are the most dangerous beans. They are flat and shining and may be either a shade of pink, purple, or marbled. They are extremely poisonous, and if given in sufficient quantity can cause death within half an hour, due to the production of hydrocyanic acid from the action of the gastric juice on the bean by its moisture and warmth. The HCN is really produced by the fermentation of a glucoside amygdalin. There is not much danger of the importation of these beans, as their properties are well known.

The Mutter Pea or Vetch and the **Riga or Dog-tooth Pea.**—These were imported in large quantities a few years ago, and produced suddenly, after being fed to horses for several weeks, most alarming symptoms, and indeed occasionally fatal results. In the stable the horse is apparently healthy, but when put to work or exertion of any kind an appalling dyspnœa or difficulty of breathing sets in, accompanied by not exactly a roaring, but rather squealing sound which may result in suffocation and death in a few minutes. It is due to a paralysis of the muscles of the larynx or entrance to the windpipe, and is of a nature very similar to that causing roaring in horses, with this remarkable exception — that roaring is an incurable disease which tends to go worse. I have never known a case of even the most severe attack of mutter poisoning which, unless death occurred as previously mentioned, did not make a complete recovery in the course of a few months. It is said that by boiling the mutters before use the poisonous property, a toxin called lathyrin, is destroyed.

Yew.—The old leaves of the yew tree, but not the seeds, are well recognized as poisonous. There are practically no symptoms in yew poisoning, collapse and death with the presence of the leaves in the stomach being all there is to be seen.

Potatoes.—Potatoes in a certain stage of putrefaction or disease undoubtedly at times develop a poison. Not much is understood at present of the cause, but pigs, which are often the recipients of diseased or brown and rotten potatoes, are often killed by them, the only post-mortem appearance being a slight congestion of the stomach. It is apparently a species of ptomaine poisoning.

Feeding Cakes

These are highly concentrated foods produced from various seeds by the removal of a portion of the oil, the residue being pressed into cake. They are prepared by " hot pressure," " cold pressure," or by the action of " solvents " which are afterwards recovered to be used again. Generally speaking, cakes are sold guaranteed to contain a certain percentage of oil, usually round about 10 per cent, with the exception of the undecorticated cotton cake which often contains no more than 6 per cent or 7 per cent of oil.

The most valuable feeding-cake is the **linseed cake.** It is the most digestible, and has slightly laxative properties, and can be given to animals of any age. Though the cakes are not considered a horse food, and are principally used for cattle and sheep, the linseed cake ground into dust is largely used for fattening horses for show purposes.

Cotton cake, obtained from the cotton seeds, is also rightly considered a most valuable cake. There are two varieties—the undecorticated and the decorticated. In the latter, the husks have been removed from the seeds. The decorticated cake is the more valuable, as the husks have a considerable irritating effect on the walls of the digestive tract, but neither variety should be used as a food for young animals, especially calves, to which it is almost a poison. In the case of cows, 2 lbs. weight per diem mixed with other foods is a sufficient supply. It is highly nitrogenous, containing over 40 per cent of proteid.

Rape cake and **Palm nut** cake are also used in some parts of the country, but in my experience cattle are not very fond of them. The Soya bean cake I have mentioned previously (p. 531).

The composition of these cakes is as follows:

	Linseed Cake.	Undecorticated Cotton Cake.	Decorticated Cotton Cake.	Rape Cake.	Palm Nut Cake.	Soya Bean Cake.
Water .	11·8	10·6	8·9	10·4	10·2	15·60
Proteids .	28·7	24·7	43·6	30·7	16·1	41·37
Fat . . .	10·7	6·6	14·9	9·8	9·5	8·60
Carbohydrates	32·1	26·0	19·7	30·1	41·9	24·81
Cellulose . .	9·4	24·9	5·7	11·3	18·3	4·37
Salts	7·3	7·2	7·2	7·7	4·0	5·25

There is also a number of **compound** or **mixed cakes**—proprietary cakes made by different firms, many of them very good, but, of course, of varying composition, and more or less secret, and often containing a spice which perhaps may add to the amount digested.

Adulteration.—All feeding-cakes lend themselves to adulteration, and should only be bought from a reputable firm. The legal definition of adulteration or falsification (which word I much prefer as conveying the meaning more plainly) includes the following conditions :

1. If any valuable ingredient naturally present has been extracted.

2. If a less valuable ingredient has been substituted.

3. If it be coloured, powdered, or polished with intent to deceive or to make the article appear of better quality than it really is.

4. If it be a substitute or imitation of a genuine article, and offered under the name of that article.

An example of the first-named condition is occasionally found in crushed oats from which a portion of the meal has been extracted during the process of crushing, the result being exactly as if some empty oat husks had been added. Perhaps the commonest adulteration of cakes is to mix a cheaper cake with a more valuable one, thus, rape is often mixed with linseed. Except that it is fraud this is not harmful, but cakes are also mixed with deleterious materials, the refuse husks of the castor or even the croton bean being sometimes found, particularly in the cotton cakes. I have also found quantities of chopped string and sacking as an adulteration. The detection of these frauds is not always very easy owing to the finely-powdered condition of the adulterant in the mixture. A magnifying glass is useful, but often nothing short of a microscopical examination by an expert, who can detect from the appearance of the starch grains the plant from which they are derived, is sufficient to determine what the adulterant is.

Roots, etc.

The principal roots and allied foods are turnips, swedes, mangel-wurzel, kohl-rabi, cabbage, potatoes, and carrots. They are especially winter and early spring diets, and are very useful as supplying fresh vegetable material until grass time. They consist of 80 per cent to 90 per cent of water, the balance being made up mostly of carbohydrate.

Turnips and swedes are used for horses, cattle, and sheep. They may be given whole, but should never be given sliced to horses or cattle, the top slice, particularly in the case of cattle, being a frequent cause of choking. A cow does very little grinding of her food when she is hungry, but bolts it and depends upon chewing her cud for proper mastication. The top slice often becomes fixed in the throat or upper portion of the œsophagus, where it is very difficult to deal with, the sharp edge injuring and sometimes even penetrating the delicate wall of the œsophagus with fatal results. They should always either be cut into fingers or pulped. A cow or bullock may be allowed up to half a hundredweight of turnips per day.

Mangel-wurzel is a species of beet, and is better buried in the ground until the early spring when the turnips are finished, as there is a carbohydrate named pectin which becomes changed into a more digestible sugar in the course of two or three months after pulling. Mangels (or mangolds) should only be used very sparingly to sheep, as in them they are apt to produce urinary disease if given in quantity.

Kohl-rabi and **cabbage** are quite healthy foods and do not tend to taint the milk to the extent that turnips do, though turnips do not taint the milk much if they are given after or during the process of milking, and not shortly before.

Potatoes are not a good horse food in any quantity, and unless given boiled are apt to produce purging and colicky pains. A few raw sliced potatoes to a horse sick from indigestion are often very useful, they stimulate the salivary glands and sweeten a foul mouth, but half a dozen are quite sufficient, more are likely to do harm. Cooked they may be used in fair quantity for cattle, but the small unmarketable potatoes are best used for feeding pigs. Uncooked potatoes should never be given whole to either cattle or horses. They are a not uncommon cause of choking in both.

Carrots are a most valuable food for either horses or cattle, but are usually too expensive to give to the latter. They contain more digestible proteïd than the other roots, and are very useful for either healthy or sick horses, as also for young colts, by all of which they are much appreciated. They are laxative and slightly diuretic. Carrots should always be cut lengthwise or finely chopped, otherwise they, too, may *be a cause of choking.*

Cooking Foods.

Steaming or cooking of food is only advisable where fattening is the object to be obtained, or in the case of colts two to four years of age when the gums are tender from the irruption of the molar teeth, or again in the case of old animals with defective teeth.

Dirt and Dust Extraction.

In all stables where the number of horses is considerable, a dust extractor is most desirable and economical, the amount of dust extracted being astonishing.

Horses should as a rule be fed by weight and not by bulk. Oats, for example, will vary as much as 20 per cent to the bushel.

It is a useful rule, if there is a weighing machine available, that working horses should be regularly weighed, say once a month, and the weights noted for reference. In this way an early discovery can be made of a horse losing or gaining weight and its cause sought for, particularly as to overwork, over, under, or improper feeding, or the presence of disease. In any of these cases it is obviously to the advantage of the owner that he should as soon as possible become acquainted with the fact.

W. W.

WATER

About 64 per cent of the animal body consists of water. Its necessity and importance as an article of diet is therefore obvious. Water has the definite chemical composition of 16 parts by weight of oxygen and 2 parts by weight of hydrogen, and is represented by the chemical formula H_2O. It has one peculiar characteristic which is probably unique, *i.e.* while the law with all other solids, liquids, or gases is that they expand with heat and contract with cold, with water it is not so. Water has its greatest density at 4° centigrade or 39·2° Fahrenheit, and whether heated from this temperature upwards or cooled downwards it expands in both cases. If this were not so when water was cooled below 4° C. the cooler water being heavier from contraction would sink to the bottom and you would then get ice formed from the bottom upwards, whereas, as we know, ice is formed on the top of the water first. The quantity of water required by an adult man for drinking purposes, exclusive of that contained in his solid food, is from 50 to 60 ounces daily. A horse requires 6 to 7 gallons, cows about the same, elephants 25 gallons, camels 10 gallons, pigs 1 gallon. Sheep rarely drink, as generally speaking the amount of water contained in the food is sufficient. They will drink, however, during a dry season.

All natural water is derived from rain, which, falling on the earth, flows in the form of streams and rivers, some reaching the sea, some evaporating, and some sinking through cracks and fissures of the earth until it reaches an impermeable stratum, where it forms the ground water and flows to the nearest water channel or finds its way to the surface in the form of springs.

Rain-water, as it falls in the country, is pure and wholesome. It is soft owing to the absence of lime and magnesium salts.

A simple method of calculating the amount of water given by rain is to multiply the area in square feet of the receiving surface by half the rainfall in inches, the result is in gallons; for example, 1 inch of rain on 1 square yard is 1 inch on 9 square feet, which divided by 2 equals 4½ gallons for each inch of rain on 1 square yard of land. This is not quite correct, but near enough for practical purposes. One inch of rain delivers 101 tons by weight or 22 617 gallons on each acre. If the inches of rain be multiplied by 14½, the result equals the millions of gallons per square mile, *i.e.* 1 inch of rain equals 14½ millions of gallons per square mile.

Well-water.—Wells are divided into shallow, deep, and artesian. *Artesian wells* are wells of great depth, so called from having been first sunk in the province of Artois in France, and are usually bored for the purpose of getting a large and constant supply of water. A *shallow well* has been defined as a well under 50 feet in depth, unless it passes through an impervious stratum, when it is classed as a *deep well.* This is an unsatisfactory definition. A shallow well should be defined as a well capable of being contaminated by surface drainage, and *per contra* a deep well one which, deriving its water from below an impervious layer, is not likely, if properly constructed, to receive such contamination.

In making a well certain points should be borne in mind. The wall or shaft of the well should extend a foot or so above the ground to avoid surface flooding. It should be impervious from the top down to the first impermeable layer, and should be covered to prevent contamination from dead leaves, birds, etc. Deep well water is usually good, but no such water ought to be used for drinking purposes unless previously subjected to a chemical analysis.

The *characteristics of a pure water* are absence of colour, taste, smell, or extreme hardness; but these are only general rules, a spring water may be very impure from metallic salts, and a bright-looking water may be charged with lead, whereas a rusty-coloured water may contain nothing worse than oxide of iron or rust. Some wells are known for their medicinal properties. Altogether, it may be repeated that the only reliable tests are chemical analysis and microscopic and bacteriological examinations.

The *impurities found in water* are divided into organic and inorganic. The organic are animal and vegetable substances in all forms, from disease-producing organisms to harmless vegetable growths. Living vegetation is usually harmless, but decomposing vegetable matter is objectionable. A useful plan for deciding whether vegetable matter is harmful is to fill a bottle with water and leave it stoppered for a few days in a warm place, when, if decomposition is going on, it can be noticed by the smell on removing the cork.

The effects of drinking impure water are not so well marked or so numerous in the lower animals as in human beings, in whom it is well recognized many epidemics may be caused, such as cholera, enteric, typhoid, dysentery, dyspepsia, diarrhœa, goitre, and others. The lower animals do not suffer from most of these complaints, but on the other hand the diseases from which they do suffer, such as glanders, cattle plague, foot and mouth disease, epizoötic abortion, anthrax, and probably tuberculosis, may be conveyed by water, as also the intestinal and other parasites which are so frequent in the lower animals.

The organic impurities which especially point to a water being dangerous to human beings are those which point to the presence of sewage or decomposing animal matter, and since we may consider that the water to which sewage can gain access is also likely to be contaminated with surface refuse of all kinds, we also accept the presence of sewage matter as constituting a dangerous water for animals. In testing for sewage the chemist analyses for certain salts which the decomposition of animal excreta produces in water, and the combined presence of ammonia, chlorides, nitrites, nitrates, and phosphates may be taken as absolutely conclusive evidence of sewage contamination. The presence of chlorides alone might only mean the presence of common salt, probably due to the water passing through a brine or salt-bearing stratum. Nitrites are very important; they point to the contamination being recent, as nitrites soon become oxidized into nitrates. The presence of nitrates without nitrites would point to an old contamination which was not going on at the time. Phosphates are indicative of contamination from urine, especially if ammonia salts are also present.

Microscopical examination of water is chiefly valuable in discovering the presence of the ova of parasites, but might point to the presence of sewage if epithelial cells or shreds of muscular fibre were found.

Bacteriological examination is now considered the most reliable method and is almost universally carried out in testing for sewage, Koch's plate culture being the process adopted, the media being generally gelatine agar and bile-salt agar, which latter seems to have an inhibitory action on the growth of those germs which may be said to be non-fæcal, while *B. typhosus* and *B. coli communis* develop a characteristic appearance.

All water taken for analysis should be dealt with rapidly or else kept at freezing-point, otherwise the germs originally present will multiply and thus give a false estimate.

Hard and Soft Water.—The hardness of water is caused by the presence of metallic salts in solution. Some are in true solution, others are only held in solution by CO_2. In the first group, sulphates and chlorides of calcium and magnesium are the cause of the hardness, and as boiling the water does not rid it of this the condition is known as *permanent hardness*. In the second group, the carbonates of calcium and magnesium are held in solution as bicarbonates by the CO_2 present in the water. Boiling in this case drives off the CO_2 and then the carbonates are precipitated. The condition set up by these carbonates is known as *temporary hardness*. Another method of softening temporarily hard water on a large scale is to add lime-water, which combines with the carbonic acid in the water, and thus removes the solvent, the whole of the lime being then precipitated as $CaCO_3$ (chalk). The chemical equation would be $CaCO_3 + CO_2 + CaH_2O_2 = 2\ CaCO_3 + H_2O$.

Methods of Detection.—(1) Boiling the water, collecting and weighing the carbonates will show how much temporary hardness is present but gives no indication of the extent of the permanent hardness.

(2) Soap added to hard water causes the precipitation of all the organic salts as insoluble stearates and oleates. On this fact is based a very useful method of detecting this impurity. B.P. soap liniment is used, and it is found that 1 minim added to half an ounce of water is equivalent to $1\frac{1}{2}$ grains of lime per gallon, *i.e.* soap liniment is added drop by drop in a test-tube and the water shaken until lather commences to form. One first estimates the total hardness of water by this method, and then taking a fresh sample and boiling proceeds to estimate the permanent hardness. In this way the proportion of temporary and permanent hardness can be calculated. Water is not classed as hard up to the amount of 10 grains per gallon, after which each grain is known as one degree of hardness; thus a water containing 15 grains of lime per gallon would be described as possessing 5 degrees of hardness. Very hard water is said to be a cause of chronic indigestion, and to predispose to calculi both urinary and intestinal. It has even been blamed for the production of exostoses, and perhaps, as I have already stated, if given with food rich in salts, may be one of the causes. The best natural example of soft water is rain before it reaches the earth and therefore before it has had an opportunity of meeting with any salts. This is often collected in cisterns and used for washing purposes. The lower animals, especially horses, very much appreciate rain water. If used for collecting drinking water cisterns should be made of iron, slate, stone, or cement; a leaden cistern is a distinct danger, as soft water has an especially plumbo-solvent action and lead poisoning may easily be produced.

Lead Poisoning may be either acute or chronic. Acute lead poisoning is rarely seen except in cattle and is then due not to water but to the animals having had access to lead paint, which has a sweetish taste and is very attractive to cattle. In the chronic form it occurs in both horses and cattle, oftenest perhaps in the neighbourhood of iron and steel works where a smoke is emitted from the furnaces containing the oxides of lead and zinc, which settle on the herbage or on the window sills of stables where both horses and cattle will lick it with evident enjoyment. Chronic lead poisoning from water impregnated with the salts of lead is not very easily diagnosed. One is usually led to the diagnosis from the fact of several horses or cattle being affected in the same way. The chief symptoms are: appetite capricious, loss of flesh, and in the case of horses, stumbling over their work and ultimately in their walk; a somewhat blue appearance of the gums, though I have never been able to distinguish the blue line over the teeth said to be characteristic of lead poisoning in human beings and to be due to the combination of lead with the tartar of the teeth. In horses the symptoms might be mistaken for rheumatism and in cattle for tuberculosis, with a tendency in both horses and cattle to knuckle over at the fetlocks. In case of suspicion of lead poisoning not only the cistern should be inspected but, particularly in old buildings from the roofs of which the water is gathered, an examination should be made of the spouts, which in the old times were often lined with lead. If there is a reasonable suspicion, then of course a chemical examination will settle the matter.

Water from a peaty district is also plumbo-solvent from the presence of humic acid, but this water under ordinary circumstances is not likely to have the opportunity of exercising this function.

Generally speaking, a lead pipe used as a service pipe is not dangerous if the supply is continuous, the carbonic acid in the water combining with the lead to form a fine coating of insoluble lead carbonate on the inside of the pipe, and so long as air is excluded it is quite safe, but if the pipes are emptied, as in an intermittent supply during a water famine, the action of the air is to reduce the insoluble carbonate into a soluble oxide, and so the water may become dangerous.

Purification of Water.—Nowadays nearly every village has an ample supply of good water, and filtration for its purification by means of house filters is rarely necessary. It is never to be recommended, as there is no such thing as a good house filter. Animal charcoal, of which many filters are composed, actually favours the development of micro-organisms, and unless frequently renewed does more harm than good.

Deep sand and gravel are the only perfect filters. They not only hold back the suspended matter, but the sand has also the power of holding back micro-organisms, that is, provided the layer of sand is not less than one foot thick and that the water be not forced through it at a greater rate than four inches per hour.

Alum has a great power of purifying water from suspended matter; it produces a film which carries down with it the suspended matter and clarifies water to a remarkable degree. The quantity added is about six grains per gallon, and it is largely used by mineral water manufacturers to clarify and brighten the water, but it does not to any extent remove the micro-organisms, and this is now considered the most important part of water purification.

To take a sample of water for analysis certain care must be taken. Winchester quart bottles holding about half a gallon are very suitable; the bottle used should be carefully cleansed with a little weak hydrochloric acid and then thoroughly washed out with some of the water to be examined. When a sample is taken the bottle should be filled quite full and then a little poured out to allow for the glass stopper. The bottle during filling should be slowly immersed in the water to a few inches below the surface, so that surface water does not run into it, and then as mentioned before it should be dealt with quickly or otherwise kept at freezing-point.

W. W.

A CLASSIFIED LIST OF WORMS AFFECTING THE DOMESTICATED ANIMALS

Horse.

TREMATODES

		Common Habitat.
Fascioloidea.		
Fasciola.	*F. hepatica*, Linnæus 1758. Syn. *Distomum hepaticum.*	Liver. Bile ducts.
	F. gigantica, Cobbold 1856. Syn. *F. augusta.*	” ”
Schistosoma.	*S. indicum*, Montgomery 1905. Syn. *Bilharzia indica.*	Portal veins.
Paramphistomoidea.		
Pseudodiscus.	*P. collinsi* (Cobbold 1875). Syn. *Amphistoma collinsi.*	Large intestine.
Gastrodiscus.	*G. ægyptiacus* (Cobbold 1876).	Small intestine.
	G. secundus, Looss 1907, in the Mule in Assam.	” ”

CESTODES

Anoplocephalidæ.		
Anoplocephala.	*A. magna* (Abildgaard 1789). Syn. *T. plicata.* Syn. *Taenia magna.*	Cæcum and colon.
	A. perfoliata (Goeze 1782). Syn. *T. perfoliata.*	” ”
	A. mamillana (Mehlis 1831). Syn. *T. mamillana.*	Small intestine.
Tæniidæ (Larval stages only).		
	Cœnurus cerebralis (Batsch 1786), larval stage of *Multiceps multiceps* (Laske 1780).	Cranium.
	Cysticercus tenuicollis (Rudolphi 1810), larval stage of *T. hydatigena,* Pallas 1766.	Peritoneum.
	Echinococcus (or Hydatid), larval stage of *Echinococcus granulosus* (Batsch 1786).	Lungs and liver.

NEMATODES

Ascaridæ.		
Ascaris.	*A. megalocephala,* Cloquet 1824.	Intestines (mainly small).
Oxyuridæ.		
Oxyuris.	*O. equi,* Schrank 1788. Syns. *Oxyuris curvula, O. mastigodes*	Large intestine (colon and rectum).
Probstmayria.	*P. vivipara* (Probstmayer 1865).	Colon.

Horse (*contd.*).

NEMATODES (*contd.*)

		Common Habitat.
DRACUNCULIDÆ.		
Dracunculus.	*D. medinensis* (Linnæus 1758).	Subcutaneous tissues of limbs.
FILARIIDÆ.		
Filaria.	*F. cordicola*, v. Linstow 1905.	
	F. hæmorrhagica.	Skin and subcutaneous tissues.
	Syn. *F. multipapillosa.*	
	F. herpetica.	,, ,, ,,
Dermofilaria.	*D. irritans*, Rivolta 1884.	,, ,, ,,
	Syn. *Filaria irritans.*	
Setaria.	*S. equina*, Abildgaard 1789.	Peritoneum.
	Syn. *F. papillosa.*	
	S. labiato-papillosa (Alessandrini 1838).	,,
	Syn. *F. cervina.*	
Onchocerca.	*O. reticulata*, Diesing 1851.	Muscular and tendinous tissues.
	O. cervicalis, Railliet and Henry 1910.	Neck (ligamentum nuchæ).
Thelazia.	*T. lachrymalis* (Gurlt 1831).	Orbit.
SPIRURIDÆ.		
Habronema.	*H. megastoma* (Rudolphi 1819).	Gastric mucous membrane.
	Syn. *Spiroptera megastoma.*	
	H. microstoma (Rudolphi 1819).	,, ,, ,,
	Syn. *Spiroptera microstoma.*	
Gongylonema.	*G. scutatum* (Müller 1869).	Œsophagus and pharynx.
	Syn. *Spiroptera scutatum.*	
STRONGYLIDÆ.		
Strongylus.	*S. equinus*, Müller 1784.	Cæcum and colon.
	S. vulgaris (Looss 1900).	Cæcum and colon (larvae in aneurisms).
	S. edentatus (Looss 1900).	Cæcum and colon.
Triodontophorus.	*T. minor* (Looss 1900).	,, ,,
	T. intermedius, Sweet 1908.	,, ,,
	T. serratus (Looss 1900).	,, ,,
	T. tenuicollis, Boulenger 1916.	,, ,,
	T. brevicauda, Boulenger 1916.	,, ,,
Gyalocephalus.	*G. capitatus*, Looss 1900.	
Œsophagodontus.	*O. robustus* (Giles 1892).	
Cylindropharynx.	*C. brevicauda*, Leiper 1911.	
	C. longicauda, Leiper 1911.	
Cylicostomum.	*C. tetracanthum* (Mehlis 1831).	,, ,,
(Syn. *Cylichnostomum*).	Syns. *Trichonema arcuata*, *Sclerostoma tetracanthum.*	,, ,,
	C. labratum, Looss 1900.	,, ,,
	C. labiatum, Looss 1900.	,, ,,
	C. coronatum, Looss 1900.	,, ,,
	C. bicoronatum, Looss 1900.	,, ,,
	C. poculatum, Looss 1900.	,, ,,
	C. calicatum, Looss 1900.	,, ,,
	C. alveatum, Looss 1900.	,, ,,
	C. catinatum, Looss 1900.	,, ,,
	C. nassatum, Looss 1900.	,, ,,
	C. radiatum, Looss 1900.	,, ,,
	C. elongatum, Looss 1900.	,, ,,
	C. auriculatum, Looss 1900.	,, ,,
	C. goldi, Boulenger 1916.	,, ,,
	C. insigne, Boulenger 1916.	,, ,,
	C. euproctus, Boulenger 1916.	,, ,,
METASTRONGYLIDÆ.		
Trichostrongylus.	*T. axei* (Cobbold 1886).	Stomach wall.
Dictyocaulus.	*D. arnfieldi* (Cobbold 1884).	Lung tissue.
	Syn. *Strongylus arnfieldi.*	
EUSTRONGYLIDÆ.		
Dioctophyme.	*D. renale* (Goeze 1782).	Kidney.

Cattle.

TREMATODES

FASCIOLOIDEA.		
Fasciola.	*F. hepatica*, Linnaeus 1758.	Bile ducts.
	Syn. *Distomum hepaticum.*	,, ,,
	F. gigantica, Cobbold 1856.	,, ,,
	Syn. *F. augusta.*	
	F. magna (Bassi 1875).	,, ,,
Dicrocœlium.	*D. hospes*, Looss 1907.	,, ,,

Cattle (*contd.*).

TREMATODES (*contd.*)

		Common Habitat.
Eurytrema.	*E. pancreaticum* (Janson 1889).	Pancreatic ducts.
	E. cœlomaticum (Giard and Billet 1892).	,, ,,
Schistosoma. Syn. *Bilharzia.*	*S. bovis* (Sonsino 1876). Syns. *Bilharzia bovis, B. crassa.*	Portal veins and urinary organs.
	S. bomfordi, Montgomery 1906.	,, ,,
	S. japonicum, Katsurada 1904.	,, ,,
	S. spindalis, Montgomery 1906.	,, ,,
	S. turkestanicum, Skrjabin 1913.	,, ,,
PARAMPHISTOMOIDEA.		
Paramphistomum.	*P. cervi* (Schrank 1790). Syn. *Amphistomum conicum.*	Rumen.
	P. tuberculatum (Cobbold 1875).	
	P. scoliocœlium, Fishoeder 1904.	
	P. epiclitum, Fischoeder 1904.	
	P. fraternum, Stiles and Goldberger 1910.	
Cotylophoron.	*C. cotylophorum,* Fischoeder 1901.	
	C. indicum, Stiles and Goldberger 1910.	
Stephanopharynx.	*S. compactus,* Fischoeder 1901.	
Homalogaster.	*H. philippinensis,* Stiles and Goldberger 1910.	
	H. poirieri, Giard and Billet 1892.	
Carmyerius.	*C. gregarius,* Looss 1896.	
	C. spatiosus, Brandes 1898.	
	C. mancupatus, Fischoeder 1901.	
Fischoederius.	*F. elongatus* (Poirier 1883).	
	F. cobboldi (Poirier 1883).	
	F. siamensis, Stiles and Goldberger 1910. Syn. *Paramphistomum siamense*	Bile ducts of calves.
Gastrothylax.	*G. ægyptiacus,* Looss 1896.	
	G. compressus, Brandes 1898.	
	G. crumenifer, Creplin 1847.	

CESTODES

ANOPLOCEPHALIDÆ.		
Moniezia.	*M. alba* (Perronceto 1879).	Intestines.
	M. benedeni (Moniez 1879).	,,
	M. denticulata (Rudolphi 1810).	,,
	M. expansa (Rudolphi 1810).	,,
	M. planissima, Stiles and Hassall 1892.	,,
Thysanosoma.	*T. giardi* (Rivolta 1878).	,,
Avitellina.	*A. centripunctata,* Rivolta 1874.	,,
Stilesia.	*S. globipunctata,* Rivolta 1874.	,,

TAENIIDÆ (larval stages only).	
Cysticercus bovis, Cobbold 1866, larval stage of *T. saginata* (Goeze 1782).	Muscle tissue.
Cysticercus tenuicollis (Rudolphi 1810), larval stage of *T. hydatigena,* Pallas 1766.	Peritoneum.
Cœnurus cerebralis (Batsch 1786), larval stage of *Multiceps multiceps* (Leske 1780).	Cranium.
Echinococcus (or Hydatid), larval stage of *Echinococcus granulosus* (Batsch 1786).	Lungs and liver.

NEMATODES

ASCARIDÆ.		
Ascaris.	*A. vitulorum* (Goeze 1782).	Small intestine.
FILARIIDÆ.		
Eleophora.	*E. pœli* (Vryburg 1897).	Parasitic aneurisms.
Setaria.	*S. labiato-papillosa* (Alessandrini 1838). Syn. *F cervina.*	Peritoneum.
Onchocerca.	*O. gibsoni* (Cleland 1910). Syn. *O. lienalis.*	Intermuscular connective tissue.
	O. armillata, Railliet and Henry 1909.	Aneurisms.
	O. gutterosa, Neumann 1910.	Aorta.
Thelazia.	*T. rhodesi* (Desmarest 1827).	
	T. gulosa, Railliet and Henry 1910.	
	T. alfortensis, Railliet and Henry 1910.	
DRACUNCULIDÆ.		
Dracunculus.	*D. medinensis,* Linnæus 1758.	Subcutaneous tissues of limbs.
SPIRURIDÆ.		
Gongylonema.	*G. scutatum* (Müller 1869).	Œsophagus.
	G. verrucosum (Giles 1892).	,,
GNATHOSTOMIDÆ.		
Gnathostoma.	*G. hispidum,* Fedtschenko 1839.	

Cattle (*contd.*).

NEMATODES (*contd.*)

		Common Habitat.
STRONGYLIDÆ.		
Œsophagostomum.	*O. columbianum* (Curtica 1890).	Small and large intestines.
	O. radiatum (Rudolphi 1903).	Small intestine.
Chabertia.	*C. ovina* (Fabricius 1788).	Large intestine.
	Syn. *Sclerostomum hypostomum.*	
Agriostomum.	*A. vryburgi,* Railliet 1902.	
Bunostomum.	*B. phlebotomum* (Railliet 1900).	
Gaigeria.	*G. pachyscelis,* Railliet and Henry 1911.	
SYNGAMIDÆ.		
Syngamus.	*S. laryngeus,* Railliet 1899.	Larynx.
METASTRONGYLIDÆ.		
Metastrongylinæ.		
Dictyocaulus.	*D. viviparus,* Bloch 1782.	Trachea and bronchi.
	Syn. *Strongylus micrurus.*	
Trichostrongylinæ.		
Trichostrongylus.	*T. extenuatus,* Railliet 1898.	Abomasum.
	Syn. *Str. gracilis.*	
Ostertagia.	*O. ostertagi* (Stiles 1892).	Abomasum and small intestine.
Cooperia.	*C. onchophora* (Railliet 1898).	Small intestine.
	Syn. *Strongylus ventricosus.*	
	C. pectinata, Ransom 1907.	
	C. punctata (v. Linstow 1906).	
Nematodirus.	*N. digitatus* (v. Linstow 1906).	
	N. filicollis (Rudolphi 1802).	Small intestine.
	Syn. *Strongylus filicollis.*	
Hæmonchus.	*H. contortus,* Rudolphi 1802.	Abomasum.
	Syn. *Strongylus contortus.*	
EUSTRONGYLIDÆ.		
Dioctophyme.	*D. visceralis* (Gmelin 1789).	Kidney.
	Syn. *Eustrongylus visceralis.*	
TRICHOSOMIDÆ.		
Trichuris.	*T. ovis* (Abildgaard 1795).	Cæcum.
	Syns. *Trichocephalus ovis, T. affinis.*	
	T. discolor (v. Linstow 1906).	
	Syn. *Trichocephalus discolor.*	
Capillaria.	*C. bovis* (Schnyder 1906).	
TRICHINELLIDÆ.		
Trichinella.	*T. spiralis* (Owen 1835).	
	Syn. *Trichina spiralis.*	

Sheep and Goat.

TREMATODES

FASCIOLOIDEA.		
Fasciola.	*F. hepatica,* Linnaeus 1758.	Bile ducts.
	Syn. *Distomum hepaticum.*	
	F. gigantica, Cobbold 1856.	,, ,,
	Syn. *F. augusta.*	
Dicrocœlium.	*D. lanceatum,* Stiles and Hassall 1896.	,, ,,
Schistosoma.	*S. bovis* (Sonsino 1876).	Portal veins.
	Syns. *Bilharzia bovis, B. crassa.*	
PARAMPHISTOMOIDEA.		
Paramphistomum.	*P. cervi* (Schrank 1790).	Rumen.
	Syn. *Amphistomum conicum.*	
Cotylophoron.	*C. indicum,* Stiles and Goldberger 1910.	

CESTODES

ANOPLOCEPHALIDÆ.		
Moniezia.	*M. neumanni,* Moniez 1891.	Intestines.
	M. trigonophora, Stiles and Hassall 1893.	,,
	M. expansa (Rudolphi 1810).	,,
	M. alba (Perronceto 1879).	,,
	M. benedeni (Moniez 1879).	,,
	M. planissima, Stiles and Hassall 1892.	,,
Thysanosoma.	*T. giardi* (Rivolta 1878).	,,
	T. actinioides, Diesing 1834.	,,
Avitellina.	*A. centripunctata,* Rivolta 1874.	,,
Stilesia.	*S. globipunctata,* Rivolta 1874.	,,
TÆNIIDÆ (larval stages only).		
Cœnurus cerebralis (Batsch 1786), larval stages of *Multiceps multiceps,* Leske 1780.		Cranium.
Echinococcus (or Hydatid), larval stage of *Echinoccus granulosus* (Batsch 1786).		Lungs and liver.
Cysticercus ovis (Cobbold 1869), larval stage of *T. ovis* (Cobbold 1869).		Muscle tissue.

Sheep and Goat (*contd.*).

NEMATODES

		Common Habitat
ASCARIDÆ.		
Ascaris.	*A. lumbricoides* (Linnæus 1758). Syn. *A. ovis.*	Small intestine.
FILARIIDÆ.		
Onchocerca.	*O. capræ* (v. Linstow 1883).	Tongue of goat.
ANGIOSTOMIDÆ.		
Strongyloides.	*S. papillosus* (Wedl 1856).	
SPIRURIDÆ.		
Gongylonema.	*G. scutatum* (Mueller 1869).	Œsophagus.
	G. verrucosum (Giles 1892).	,,
STRONGYLIDÆ.		
Chabertia.	*C. ovina* (Fabricius 1788). Syn. *Sclerostomum hypostomum.*	Large intestine.
Bunostomum.	*B. trigonocephalum* (Rudolphi 1808). Syn. *Uncinaria cernua.*	Intestine.
Œsophagostomum.	*O. columbianum* (Curtice 1890).	Large intestine.
	O. venulosum (Rudolphi 1809).	
METASTRONGYLIDÆ.		
Metastrongylinæ.		
Dictyocaulus.	*D. filaria* (Rudolphi 1809). Syn, *Strongylus filaria.*	Trachea and bronchi.
Metastrongylus.	*M. elongatus* (Dujardin 1845). Syn. *Strongylus.*	
Synthetocaulus.	*S. rufescens* (Leuckart 1865).	Bronchioles and lung tissue.
	S. capillaris (Müller 1889).	Lung tissue.
	S. unciphorus, Railliet and Henry 1907.	
	S. ocreatus, Railliet and Henry 1907.	
Trichostrongylinæ.		
Hæmonchus.	*H. contortus* (Rudolphi 1803). Syn. *Strongylus contortus.*	Abomasum.
Ostertagia.	*O. circumcinata* (Stadelmann 1894). Syn. *Strongylus cervicornis.*	,,
	O. trifurcata, Ransom 1907.	
	O. marshalli, Ransom 1907.	
	O. occidentalis, Ransom 1907.	
Cooperia.	*C. curticei* (Railliet 1893).	
	C. onchophora (Railliet 1898). Syn. *Strongylus ventricosus.*	Small intestine.
Nematodirus.	*N. filicollis* (Rudolphi 1802). Syn. *Strongylus filicollis.*	,, ,
Trichostrongylus.	*T. instabilis* (Railliet 1893).	
	T. probolurus (Railliet 1896).	
	T. vitrineus, Looss 1905.	
	T. extenuatus (Railliet 1898).	
	T. capricola, Ransom 1907.	
TRICHOSOMIDÆ.		
Trichuris.	*T. ovis* (Abildgaard 1795). Syn. *Trichocephalus affinis.*	Cæcum.
Capillaria.	*C. brevipes*, Ransom 1911.	
	C. longipes, Ransom 1911.	

Pig.

TREMATODES

FASCIOLOIDEA.		
Fasciolopsis.	*F. buski* (Lankaster 1857). Syn. *Distomum crassum.*	Duodenum.
Paragonimus.	*P. westermanni* (Kerbert 1878).	Lungs.
Agamodistomum.	*A. suis*, Stiles 1898.	Muscles.
PARAMPHISTOMOIDEA.		
Gastrodiscoides.	*G. hominis* (Lewis and McConnell 1876). Syn. *Gastrodiscus hominis.*	Cæcum.

CESTODES

ANOPLOCEPHALIDÆ.		
Thysanosoma.	*T. giardi* (Rivolta 1878).	Intestines.
TÆNIIDÆ (larval form only).		
Cysticercus cellulosæ, larval form of *T. solium*, Linnæus 1758.		Muscle tissue.
Cysticercus tenuicollis, larval stage of *T. hydatigena.*		Liver and peritoneum.

Pig (*contd.*).

NEMATODES

		Common Habitat.
ASCARIDÆ.		
Ascaris.	*A. lumbricoides,* Linnæus 1758. Syn. *A. suis.*	Small intestine.
ANGIOSTOMIDÆ.		
Strongyloides.	*S. suis* (Lutz —— ? n.v.).	
GNATHOSTOMIDÆ.		
Gnathostoma.	*G. hispidum,* Fedtschenko 1839.	Stomach.
TRICHINELLIDÆ.		
Trichinella.	*T. spiralis,* Owen 1835. Syn. *Trichina spiralis.*	Intestines (adult form). Muscle tissue (larval form).
TRICHOSOMIDÆ		
Trichuris.	*T. suis* (Schrank 1788). Syns. *T. crenatus* and *Trichocephalus crenatus.*	Cæcum.
FILARIIDÆ.		
Setaria.	*S. bernardi,* Railliet and Henry.	Peritoneum.
SPIRURIDÆ.		
Gongylonema.	*G. scutatum* (Müller 1869).	Œsophagus and pharynx.
	G. pulchrum, Molin 1857.	
Arduenna.	*A. strongylina* (Rudolphi 1819).	
	A. dentata (v. Linstow 1904).	
Physocephalus.	*P. sexalatus* (Molin 1860).	
Simondsia.	*S. paradoxa* (Cobbold 1864).	Stomach.
STRONGYLIDÆ.		
Œsophagostomum.	*O. dentatum* (Rudolphi 1803).	
Characostomum.	*C. longemucronatum* (Molin 1860).	
Crassisoma.	*C. urosubulatum,* Alessandrini 1909.	
Stephanurus.	*S. dentatus,* Diesing 1839. Syn. *Sclerostoma pinguicola.*	Renal fat, kidneys, liver.
Strongylus.	*S. rubidus,* Stiles and Hassall, 1892.	Stomach.
METASTRONGYLIDÆ.		
Metastrongylus.	*M. apri* (Gmelin 1791). Syns. *M. paradoxus, Strongylus paradoxus.*	Trachea and bronchi.
	M. brevivaginatus, Railliet and Henry 1897.	,, ,,
EUSTRONGYLIDÆ.		
Dioctophyme.	*D. visceralis* (Gmelin 1789). Syn. *Eustrongylus gigas.*	Kidney.

ACANTHOCEPHALA

GIGANTORHYNCHIDÆ.		
Gigantorhynchus.	*G. hirudinaceus* (Pallas 1781). Syns. *G. gigas, Echinorhynchus gigas.*	Duodenum.

Dog.

TREMATODES

HOLOSTOMOIDEA.		
Hemistoma.	*H. alatum* (Goeze 1782).	Intestines.
FASCIOLOIDEA.		
Paragonimus.	*P. westermanni* (Kerbert 1878).	Lungs.
Echinostoma.	*E. perfoliatum,* von Ratz 1908.	Intestines.
Isthmiophora.	*I. melis,* Luhe 1909. Syn. *E. trigonocephalum.*	,,
Opisthorchis.	*O. noverca,* Braun 1903.	Bile ducts.
Clonorchis.	*C. sinensis* (Cobbold 1875).	,, ,,
Metorchis.	*M. truncatus* (Rudolphi 1819).	Liver.
Heterophyes.	*H. heterophyes* (v. Siebold 1852).	Intestines.
	H. dispar, Looss 1902.	
	H. æqualis, Looss 1902.	
Metagonimus.	*M. yokogawai,* Katsurada 1913. Syn. *Yokogawa yokogawai.*	,,
Ascocotyle.	*A. minuta,* Looss 1899.	,,
	A. italica, Alessandrini 1906.	
Centrocestus.	*C. cuspidatus,* Looss 1896.	
Schistosoma.	*S. japonicum,* Katsurada 1904.	Portal and mesenteric veins.

CESTODES

CYCLOPHYLLIDEA.		
Mesocestoididæ.		
Mesocestoides.	*M. lineatus* (Goeze 1782). Syn. *Tænia litterata.*	Intestines.

Dog (*contd.*).

CESTODES (*contd.*)

		Common Habitat
Dipylidiinae.		
Dipylidium.	*D. caninum* (Linnæus 1758).	Intestines.
	Syns. *T. cucumerina*, *T. elliptica.*	,,
	D. sexcoronatum, von Ratz 1900.	,,
Tæniidæ.		
Tœnia.	*T. balaniceps*, Hall 1910.	,,
	T. brachysoma, Sette 1899.	,,
	T. brauni, Sette 1897.	,,
	T. hydatigena, Pallas 1766.	,,
	Syn. *Tœnia marginata.*	
	T. krabbei, Moniez 1879.	,,
	T. ovis (Cobbold 1869).	,,
	T. pisiformis (Bloch 1780).	,,
	Syn. *T. serrata.*	
Echinococcus.	*E. granulosus* (Batsch 1786).	,,
	Syn. *T. echinococcus.*	
Multiceps.	*M. gaigeri*, Hall 1916.	,,
	M. multiceps (Leske 1780).	,,
	Syn. *T. multiceps*, *T. cœnurus.*	,,
	M. serialis (Gervais 1847).	,,
	Syn. *Tœnia serialis.*	
PSEUDOPHYLLIDEA.		
Dibothriocephalus.	*D. latus* (Bremser 1819).	,,
	D. cordatus (Leuckart 1862).	,,
	D. fuscus (Krabbe 1865).	,,

NEMATODES

ASCARIDÆ.		
Belascaris.	*B. marginata* (Rudolphi 1802).	Small intestines (occasionally stomach).
	Syn. *Ascaris marginata.*	
Toxascaris.	*Tox. limbata*, Railliet and Henry 1911.	,, ,, ,,
SPIRURIDÆ.		
Spiroptera.	*S. sanguinolenta* (Rudolphi 1819).	Œsophagus and stomach and aneurisms.
TRICHOSOMIDÆ.		
Trichuris.	*T. depressiusculus*, Rudolphi 1809.	Cæcum.
	Syn. *Trichocephalus depressiusculus.*	
DRACUNCULIDÆ.		
Dracunculus.	*D. medinensis*, Linnæus 1758.	Subcutaneous tissue of limbs.
GNATHOSTOMIDÆ.		
Gnathostoma.	*G. spinigerum*, Owen 1836.	Stomach.
FILARIIDÆ.		
Filaria.	*F. recondita*, Grassi 1890.	
	F. acutiuscula, Molin 1857.	
	F. osleri, Cobbold 1879.	Lungs.
Dirofilaria.	*D. immitis* (Leidy 1856).	Right ventricle of the heart.
	Syns. *Filaria immitis*, *F. hœmatica.*	
ANCYLOSTOMIDÆ.		
Ancylostoma.	*A. caninum* (Ercolani 1859).	Small intestine.
	Syn. *Uncinaria trigonocephalum.*	
	A. ceylanicum, Looss 1911.	,, ,,
	A. brasiliense, Faria 1910.	,, ,,
Uncinaria.	*U. stenocephala* (Railliet 1864).	,, ,,
STRONGYLIDÆ.		
Hœmostrongylus.	*H. vasorum* (Baillet 1866).	Right ventricle and lung tissue
	Syn. *Strongylus vasorum.*	
EUSTRONGYLIDÆ.		
Dioctophyme.	*D. visceralis* (Gmelin 1789).	Kidney and liver.
	Syn. *Eustrongylus gigas.*	

Cat.

TREMATODES

FASCIOLOIDEA.		
Platynosoma.	*P. fastosum*, Kossack 1910.	Bile ducts.
Paragonimus.	*P. westermanni* (Kerbert 1878).	Lung.
Opisthorchis.	*O. felineus*, Rivolta 1885.	Bile ducts.
	O. pseudofelineus, Ward 1901.	,, ,,
	O. complexus (Stiles and Hassall 1894).	,, ,,

Cat (*contd.*).

TREMATODES (*contd.*)

		Common Habitat.
Metorchis.	*M. albidus* (Braun 1893).	Bile ducts.
	M. complexus, Stiles and Hassall 1894.	
	M. truncatus (Rudolphi 1819).	Liver.
Clonorchis.	*C. sinensis* (Cobbold 1875).	Gall-bladder and bile ducts.
Heterophyes.	*H. heterophyes* (v. Siebold 1852).	Intestines.
	H. dispar, Looss 1902.	,,
	H. æqualis, Looss 1902.	,,
Metagonimus.	*M. yokogawai*, Katsurada 1913.	,,
Ascocotyle.	*A. minuta*, Looss 1899.	,,
Schistosoma.	*S. japonicum*, Katsurada 1904. Syn. *Bilharzia japonicum.*	Portal and mesenteric vessels.

CESTODES

CYCLOPHYLLIDEA.		
Mesocestoididæ.		
Mesocestoides.	*M. lineatus* (Goeze 1782). Syn. *Tænia litterata.*	Intestines.
Dipylidiinæ.		
Dipylidium.	*D. œrleyi*, von Ratz 1900.	,,
	D. chyzeri, von Ratz 1897.	,,
	D. trinchesei, Railliet 1893.	,,
	D. pasqualei, Diamare 1893.	,,
	D. caninum (Linnaeus 1758). Syns. *T. cucumerina, T. elliptica.*	,,
Tæniidæ.		
Tænia.	*T. hydatigena*, Pallas 1766. Syn. *T. marginata.*	,,
	T. pisiformis (Bloch 1780). Syn. *T. serrata.*	,,
	T. tæniæformis (Batsch 1786). Syn. *T. crassicollis.*	,,
Echinococcus.	*E. granulosus* (Batsch 1786). Syn. *T. echinococcus.*	,,
PSEUDOPHYLLIDEA.		
Dibothriocephalus.	*D. latus* (Bremser 1819).	,,

NEMATODES

ASCARIDÆ.		
Belascaris.	*B. cati* (Schrank 1788). Syn. *Ascaris mystax.*	Small intestine, occasionally stomach.
OXYURIDÆ.		
Oxyuris.	*O. compar*, Leidy 1856.	Large intestine.
PHYSALOPTERIDÆ.		
Physaloptera.	*P. gemina* (v. Linstow).	
	P. præputialis, v. Linstow.	Intestines.
GNATHOSTOMIDÆ.		
Gnathostoma.	*G. spinigerum*, Owen 1836.	Stomach.
TRICHOSOMIDÆ.		
Trichuris.	*T. serratus*, v. Linstow 1879. Syn. *Trichocephalus serratus.*	Cæcum.
	T. campanula, v. Linstow 1888. Syn. *Trichocephalus campanula.*	,,
Capillaria.	*C. ærophilum*, Creplin 1839. Syn. *Trichosomum ærophilum.*	Intestines, trachea, and bronchi
	C. lineare, Leidy 1856. Syn. *Trichosomum lineare.*	
TRICHINELLIDÆ.		
Trichinella.	*T. spiralis*, Owen 1835.	,,
STRONGYLIDÆ.		
Ollulanus.	*O. tricuspis*, Leuckart 1865.	Stomach and intestines.
METASTRONGYLIDÆ.		
Synthetocaulus.	*S. abstrusus* (Railliet 1898). Syn. *Strongylus pusillus.*	(Embryos in lungs). Lung tissue.
ANCYLOSTOMIDÆ.		
Ancylostoma.	*A. ceylanicum*, Looss 1911.	Small intestine.
	A. brasiliense, Faria 1910.	,, ,,
	A. caninum (Ercolani 1859). Syn. *Uncinaria trigonocephalum.*	,, ,,

TABLE OF DOSES

♏=minim ; gr.=grain ; ʒ=drachm ; ℥=ounce ; O=pint.

	Horse.	Ox.	Sheep.	Pig.	Dog.
Acetanilid (Antifebrin).	ʒss.-i.	ʒss.-ii.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Acetum	℥i.-iii.	℥i.-viii.	ʒss.-i.	ʒi.-iv.	ʒss.-ii.
,, Scillæ	ʒi.-iv.	ʒii.-vi.	ʒss.-ii.	ʒss.-ii.	♏x.-xxx.
Acid Acetic	ʒi.-iv.	ʒi.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, ,, Dil.	℥i.-iii.	℥i.-iv.	℥ss.-i.	ʒi.-iv.	ʒss.-ii.
,, Acetylsalicyl (Aspirin)	ʒi.-iv.	ʒi.-vi.	ʒss.-i.	gr. x.-xxx.	gr. ss.-x.
,, Arsenios. (Pulv.)	gr. ii.-x.	gr. ii.-x.	gr. ss.-v.	gr$\frac{1}{8}$-$\frac{3}{4}$	gr. $\frac{1}{40}$-$\frac{1}{10}$
,, Benzoic	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. v.-x.
,, Boric	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Carbolic	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-iii.
,, Hydrochloric. Dil.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Hydrocyanic. Dil.	ʒss.-i.	ʒi.-ii.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Nitric. Dil.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Nitro-hydrochlor. Dil.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,. Phosphoric. Dil.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Salicylic	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Sulphuric. Dil.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Sulphurosum	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
,, Tannic	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. v.-x.
Aconitina	gr. $\frac{1}{40}$-$\frac{1}{20}$	gr. $\frac{1}{40}$-$\frac{1}{20}$	gr. $\frac{1}{100}$-$\frac{1}{80}$	gr. $\frac{1}{100}$-$\frac{1}{60}$	gr. $\frac{1}{600}$-$\frac{1}{100}$
Adrenalin (1-1000), (subcut.)	℥ss.-i.	℥ss.-i.	♏ii.-x.	♏ii.-x.	♏ii.-v.
Æther	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
Aloe	ʒi.-vi.	℥ss.-iss.	ʒi.-iii.	ʒi.-ii.	ʒ$\frac{1}{4}$.-i.
Aloin	ʒss.-i.	ʒi.-ii.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Alumen	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Ammon. Bromid.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Carb.	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. v.-x.
,, Chlorid.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Amyl Nitris	ʒi.-ii.	ʒi.-iii.	♏x.-xxx.	♏v.-xx.	♏ii.-x.
Anisi Fruct.	℥ss.-i.	℥ss.-iss.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
Antifebrin	ʒss.-i.	ʒss.-ii.	gr. v.-xv.	gr. v.-x.	gr. ss.-v.
Antimon. Nigrum	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
,, Tartarat.	Vermifuge ʒi.-iii.	Febrifuge ʒi.-ii.	gr. v.-xv.	Emetic gr. v.-x.	Emetic gr. i.-v.
Apomorph. Hydrochl.				gr. ss.-iss.	gr. $\frac{1}{20}$-$\frac{1}{2}$
Areca	℥ss.-ii.	ʒi.-iii.	ʒi.-iii.	ʒi.-ii.	gr. x.-lx.
Arecolin Hydrobrom.	gr. ss.-iss.	gr. ss-ii.	gr. $\frac{1}{8}$-$\frac{1}{4}$.		gr. $\frac{1}{40}$-$\frac{1}{10}$.
Argent. Nitras	gr. ii.-vi.	gr. ii.-x.	gr. ss.-ii.	gr. ss.-i.	gr. $\frac{1}{8}$-$\frac{1}{2}$
Arsenic (white)	See Acid Arsenios				
Asafetida	℥ss.-i.	℥ss.-iss.	ʒss.-ii.	ʒss.-i.	gr. v.-xx.
Aspirin	ʒi.-iv.	ʒi.-vi.	ʒss.-i.	gr. x.-xxx.	gr. ss.-x.
Atropin. Sulph.	gr. $\frac{1}{4}$-i.	ʒss.-iss.	gr. $\frac{1}{10}$-$\frac{1}{8}$	gr. $\frac{1}{80}$-$\frac{1}{10}$	gr. $\frac{1}{200}$-$\frac{1}{60}$
Barii Chlorid.	ʒss.-ii. (by mouth) gr. v.-xv. (intravenously)				
Belladonna Sol.	ʒss.-i.	ʒi.-ii.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Betanaphthol	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
Bismuth Carb.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Subnit.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Borax	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Caffeina	gr. v.-x.	gr. v.-xv.	gr. i.-v.	gr. ss.-iii.	gr. ss.-ii.
Calcii Chlorid.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Calcii Phosph.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Calumbae Rad.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Cambogia		ʒss.-i.	ʒss.-i.		gr. i.-ii.
Camphora	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. i.-v.
Cantharis	gr. v.-xv.	gr. v.-xx.	gr. v.-x.	gr. i.-v.	gr. ss.-i.
Capsici Fruct.	gr. x.-xxx.	gr. x.-xl.	gr. v.-x.	gr. i.-v.	gr. $\frac{1}{2}$-ii.
Carbo Ligni	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒss.-ii.	ʒss.-i.
Catechu	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Chloral Hydras	ʒii.-℥i.	℥ss.-iss.	ʒss.-iss.	ʒss.-i.	gr. v.-xxx.
Chloroform	ʒss.-i.	ʒi.-ii.	♏v.-xv.	♏v.-x.	♏i.-v.
Cinchonae Cortex	℥ss.-i.	ʒss.-iss.	ʒss.-iss.	ʒss.-i.	gr. x.-xxx.
Cinchonidin. Sulph	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. ii.-x.	gr. i.-v.
Cinchonin. Sulph.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. ii.-x.	gr. i.-v.
Cocainae Hydrochl.	gr. ii.-x.	gr. v.-xv.	gr. ss.-iii.	gr. ss.-ii.	gr. $\frac{1}{8}$-i
Codeinæ Phosph.	gr. x.-xl.	ʒss.-i.	gr. v.-x.	gr. i.-v.	gr. ss.-ii.
Creosotum	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
Cresol	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-iii.
Creta Prep.	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
Croton. Sem.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Cupri Sulph.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. -i.v.
				Emetic gr. x.-xxx.	gr. v.-xv.
Diamorph. Hydrochlor. (Heroin)	gr. ss.-i.				gr. -ss$\frac{1}{8}$
Digitaline (hypoderm.)	gr. $\frac{1}{6}$-$\frac{1}{2}$.	gr. $\frac{1}{4}$-i.	gr. $\frac{1}{30}$-$\frac{1}{10}$.	gr. $\frac{1}{20}$-$\frac{1}{10}$.	gr. $\frac{1}{120}$-$\frac{1}{30}$.
Digitalis Fol.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Ergota	℥ss.-i.	℥ss.-iss.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
Ergotin (Ext. Ergot.)	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
Eserine (See Physostigmine)					
Eucaine	gr. i.-x.	gr. v.-xv.	gr. ss.-iii.	gr. ss.-ii.	gr. $\frac{1}{8}$-$\frac{1}{2}$

TABLE OF DOSES—*continued.*

	Horse.	Ox.	Sheep.	Pig.	Dog.
Ext. Belladon. Vir.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. $\frac{1}{8}$-ii.
,, Cannabis Ind.	ʒi.-iv.	ʒi.-iv.	gr. x.-xxx.	gr. v.-xv.	gr. $\frac{1}{2}$-iii.
,, Cascaræ Liq.					gr. ss.-i.
,, Ergot.	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
,, Ergot. Liq.	ʒi.-ii.	ʒi.-iv.	♏x.-xxx.	♏x.-xx.	♏v.-xv.
,, Filicis Liq.	℥ss.-ii.	ʒi.-ii.	ʒi.-iii.	ʒss.-ii.	ʒss.-i.
,, Ipecac. Liq.	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏ii.-x.	♏ss.-ii.
,, Nucis Vom. Liq.	ʒss.-i.	ʒi.-iss.	♏v.-xv.	♏v.-x.	♏ss.-ii.
,, Opii Sicc.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. ii.-x.	gr. $\frac{1}{8}$-i.
Ferri Carb. (Oxid.)	ʒi.-ii.	ʒi.-iii.`	gr. x.-xxx.	gr. v.-xx.	gr. v.-x.
,, ,, Sacc.					gr. x.-xxx.
,, Sulphas	ʒss.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-v.
Galla	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Gentianæ Rad.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Glycerin	℥i.-ii.	℥i.-iii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
Glycyrrhizæ Rad.	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
Heroin (Diamorph.)	gr. ss.-i.				gr. $\frac{1}{80}$-$\frac{1}{8}$
Hexamine	ʒi.-iv.	ʒi.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Hydrarg. Iod. Rub.	gr. ii.-vi.	gr. ii.-x.	gr. ss.-i.	gr. $\frac{1}{8}$-ss.	gr. $\frac{1}{40}$-$\frac{1}{8}$
,, Perchlor.	gr. ii.-vi.	gr. ii.-x.	gr. ss.-i.	gr. $\frac{1}{8}$-ss.	gr. $\frac{1}{40}$-$\frac{1}{8}$
,, Subchlor.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
,, c̄ Creta.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Hydrogen. Perox.	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
Iodoform	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-iii.
Iodum	gr. x.-xxx.	gr. x.-xi.	gr. i.-v.	gr. i.-v.	gr. ss.-ii.
Ipecac. Rad.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
				Emetic	
				ʒss.-i.	gr. x.-xxx.
Jalapa	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Jalapæ Resina	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Kamala	℥i.-ii.	℥i.-iii.	ʒi.-iii	ʒi.-ii.	ʒss.-i.
Kerocaine (Kerfoot's novocaine)	As Cocaine				
Kino	ʒi.-iv.	ʒi.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Liq. Ammon. Acet.	℥ii.-vi.	℥ii.-x.	℥ss.-iss.	℥ss.-i.	ʒss.-iv.
,, Ammoniæ	℥ss.-i.	℥ss.-iss.	ʒss.-ii.	ʒss.-i.	♏v.-xxx.
,, Arsenicalis	℥ss.-i.	℥ss.-iss	ʒss.-iss.	♏x.-xxx.	♏i.-x.
,, Arsen. et Hydrarg. Iod.	℥ss.-i.	℥ss.-iss.	ʒss.-iss.	♏x.-xxx.	♏i.-x.
,, Calcis			℥i.-iv.	℥i.-ii.	ʒss.-iv. .
,, Ferri Perchlor.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Potassæ	℥ss.-i.	℥ss.-iss.	ʒss.-ii	ʒss.-i.	♏x.-xxx.
,, Strychnin. Hydroch.	ʒss.-ii.	ʒss.-iii.	♏x.-xx.	♏v.-x.	♏i.-iv.
Magnes. Carbonas.	ʒi.-iv. (Foals and calves)	ʒii.-iv.	ʒss.-i.	ʒss.-i.	gr. x.-xxx.
,, Sulph.	℥i.-iv.	lb. ss.-iss.	℥i.-iii.	℥ss.-i.	ʒi.-iv.
Methylditannin (See Tannoform)					
Methyl Salicylas	ʒi.-ii.	ʒi.-iii.	♏x.-xxx.	♏v.-xx.	♏v.-x.
Morphinæ Hydroch.	gr. ii.-x.	gr. v.-x.	gr. ss.-ii.	gr. ss.-iss.	gr. $\frac{1}{4}$-ii.
Myrrha	ʒi.-iv.	ʒi.-℥i.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
Naphthalin	ʒss.-ii.	ʒss.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. v.-x.
Novocaine	As Cocaine				
Nux Vomica	ʒss.-i.	ʒss.-ii.	gr. v.-xv.	gr. v.-x.	gr. ss.-iii..
Oleum Anisi	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Caryophyll.	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Chenopodii.	ʒi.-iv.	ʒi.-iv.	♏x.-xxx.	♏x.-xxx.	♏ss.-vi.
,, Croton.	♏x.-xx.	♏x.-xxx.	♏v.-x.	♏i.-iii.	♏ss.-ii.
,, Eucalypti	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Gaultheriæ	ʒi.-ii.	ʒi.-iii.	♏x.-xxx.	♏v.-xx.	♏v.-x.
,, Lini	Oss.-iss.	Oi.-ii.	℥i.-x.	℥ii.-vi.	℥ss.-iss.
,, Menth. Pip.	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Morrhuæ (Cod-liver oil)	℥ii.-viii.	℥v.-x.	℥i.-iii.	℥i.-ii.	ʒi.-iv.
,, Olivæ	Oss.-iss.	Oi.-ii.	℥ii.-x.	℥ii.-vi.	℥ss.-iss.
,, Ricini (Castor oil)	Oss.-iss.	Oi.-ii.	℥ii.-x.	℥ii.-vi.	℥ss.-iss.
,, Terebinth. (Turpentine)	℥ss.-ii.	℥i.-iii.	ʒi.-iii.	ʒss.-i.	♏v.-xv.
Opium	ʒss.-ii.	ʒss.-ii.	gr. x.-xxx.	gr. v.-xv.	gr. i.-v.
Orthoform					gr. v.-x.
Pelletierin. Tannas	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. ii.-x.	gr. i.-v.
Phenacetin	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
Physostigmin. Sulph.	gr. ss.-iss.	gr. i.-ii.	gr. $\frac{1}{8}$-$\frac{1}{4}$	gr. $\frac{1}{40}$-$\frac{1}{10}$	gr. $\frac{1}{60}$-$\frac{1}{20}$
Pilocarpin. Nitras	gr. ii.-v.			gr. $\frac{1}{4}$-i.	gr. $\frac{1}{10}$-$\frac{1}{4}$
Plumbi Acetas	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Potass. Acetas	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Bicarb.	℥ss.-i.	℥ss.-ii.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
,, Brom.	ʒi.-℥i.	℥ss.-i.	ʒss.-ii.	ʒss.-i.	gr. v.-xxx.
,, Chloras	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Iodid.	ʒss.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. v.-x.
,, Nitras	ʒss.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
,, Permang.	gr. x.-xxx.	gr. x.-xl.	gr. i.-v.	gr. i.-iv.	gr. $\frac{1}{4}$.-ii.
Pulv. Ipecac. Co.	ʒi.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.

Table of Doses—*continued.*

	Horse.	Ox.	Sheep.	Pig.	Dog.
Quininæ Hydrochlor.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
,, Sulphas	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Resina	℥ss.-i.	ʒss.-iss.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
Rhei Radix	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xx.
Salol.	ʒi.-iv.	ʒiss.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv
Santonina	gr. xv.-lx.	ʒss.-i.	gr. v.-xv.	gr. ii.-x.	gr. i.-v.
Scillæ Pulv.	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-v.
Sodii Benzoas	℥ss.-i.	℥ss.-iss.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
,, Bicarb.	℥ss.-i.	℥ss.-iss.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
,, Bromid.	ʒi.-℥i.	℥ss.-i.	ʒss.-ii.	ʒss.-i.	gr. x.-xxx.
,, Chlorid.	℥ss.-i.	℥iv.-lb. i.	℥i.-iv.		gr. x.-xxx.
,, Hyposulph.	℥ss.-i.	lb. ss.-i.	℥-i.iv.	ʒss.-ii.	gr. v.-xv.
,, Nitris	gr. x.-xxx.				gr. ss.-ii.
,, Phosphas	℥i.-iv.	℥ii.-viii.	℥ss.-iss.	℥ss.-i.	ʒss.-ii.
,, Phosphas Acid.	ʒi.-iv.	ʒii.-℥i.	ʒss.-i.	℥ss.-i.	gr. v.-xx.
,, Salicyl.	ʒi.-iv.	ʒi.-vi.	ʒss.-i.	gr. x.-xxx.	gr. v.-xv.
,, Sulphas	℥i.-iv.	lb ss.-i.	℥i.-iii.	ʒi.-℥i	ʒss.-ii.
Spirit. Ætheris	℥ss.-ii.	℥i.-iii.	ʒi.-iv.	ʒss.-ii.	ʒss.-i.
,, Ætheris Nitros.	℥ss.-ii.	℥i.-iii.	ʒi.-iv.	ʒss.-ii.	ʒss.-i.
,, Ammon. Aromat.	℥ss.-i.	℥ss.-ii.	ʒss.-ii.	ʒss.-i.	♏x.-xxx.
Stovaine	As Cocaine				
Strychnin. Hydrochlor.	gr. $\frac{1}{4}$-ii.	gr. ss.-ii.	gr. $\frac{1}{18}$-$\frac{1}{4}$	gr. $\frac{1}{30}$-$\frac{1}{20}$	gr. $\frac{1}{500}$-$\frac{1}{60}$
Sulphur Sub.	℥ss.-ii.	℥i.-iii.	ʒi.-iv.	ʒss.-iss.	gr. x.-xxx.
Syrup. Chloral					♏xv.-ʒi.
,, Ferri Iodid.					ʒss.-i.
,, Ferri Phosph. c̄ Quin. et Strychn. Easton's Syr.					♏v.-ʒss.
Syrup. Scillæ					ʒss.-i.
,, Tolu.					ʒss.-ii.
Tannoform	ʒi.-iv.	℥ss.-i.	ʒi.-ii.	ʒss.-i.	gr. v.-xv.
Tenaline					♏iss. per lb. body weight. Max. ʒi.
Thymol	gr. x.-lx.	ʒss.-iss.	gr. v.-xx.	gr. i.-v.	gr. ss.-ii.
Tinct. Aconiti	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Aconiti (Flem.)	♏v.-xv.	♏x.-xx.	♏i.-v.	♏i.-iii.	♏$\frac{1}{8}$-i.
,, Belladonnæ	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Benzoin. Co.	℥ss.-iss.	℥i.-ii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
,, Buchu	℥ss.-iss.	℥i.-ii.	ʒi.-iii.	ʒi.-ii.	♏xv.-ʒi.
,, Camph. Co.	℥ss.-iss.	℥i.-ii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
,, Cannabis Ind.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Capsici	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Catechu	℥ss.-iss.	℥i.-ii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
,, Chlorof. et Morph. Co. (Chlorodyne)	℥i.-ii.	℥i.-ii.	ʒi.-ii.	ʒss.-i.	♏v.-xv.
,, Digitalis	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Hyoscyami	℥ss.-iss.	℥i.-ii.	ʒi.-iii.	ʒi.-ii.	ʒss.-i.
,, Iodi Mitis	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
,, Nucis Vom.	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-x.
,, Opii	℥ss.-i.	℥ss.-ii.	ʒss.-ii.	♏x.-xxx.	♏v.-xv.
,, Scillæ	ʒi.-iv.	ʒii.-vi.	ʒss.-i.	♏x.-xxx.	♏v.-xv.
,, Strophanthi	ʒss.-i.	ʒss.-iss.	♏v.-xv.	♏v.-x.	♏i.-v.
Vinum Ipecac.				Expectorant Emetic	♏x.-xxx. ʒi.-iv.
Yohimbine	gr. ss.-iss.	gr. ss.-ii.			gr. $\frac{1}{100}$-$\frac{1}{10}$
Zinci Oxidum	ʒss.-ii.	ʒi.-iii.	gr. x.-xxx.	gr. v.-xx.	gr. ii.-x.
,, Sulphas	ʒss.-i.	ʒss.-iss.	gr. v.-xv.	gr. v.-x.	gr. i.-iii. Emetic gr.v.-xx.
Zingiber	℥ss.-i.	℥i.-ii.	ʒi.-iv.	ʒi.-ii.	gr. v.-xx.

B. G.

ADDENDUM

GRASS DISEASE IN THE HORSE

Grass Sickness—Forage Poisoning

For several years past a disease of horses in certain parts of Scotland and North of England has been recognized by the above names owing to the fact that it has been regarded as being due to the ingestion of food, either grass or hay, and usually appearing in the grass season. Dr. Tocher of Aberdeen, who has been carrying out investigations into the disease, suggests that the name is a misnomer, and that the condition is due to an anaerobic bacillus which he has demonstrated in the spleen and elsewhere closely resembling *Bacillus botulinus*. The symptoms resemble to some extent those produced in botulism in man. Professor Gaiger of Glasgow, however, has demonstrated in the cerebro-spinal fluid of a case sent to him an organism, which he terms a diplo-streptococcus. In his view the symptoms and post-mortem appearances of the disease resemble an affection of horses in Borna to which the name Borna disease has been given.

H. M'Vean has expressed the view that it is a nervous disease, whatever the pathology may be ultimately proved to be, and this is confirmed by the apparent paralysis of the bowel, the twitchings of the muscles, the difficulty in swallowing, and the convulsive spasms of retching.

The etiology of the disease is at the present time, however, not satisfactorily demonstrated, though opinions appear to be divided between the two afore-mentioned claims, and one or other may ultimately prove to be correct. In either case it is more than likely that the term "Grass disease," by which it is already so well known to veterinarians, will remain.

By many observers the disease is regarded as affecting horses mainly in the prime of life (from 3 to 10 years), though cases are known to occur at any age. In the view of Mr. W. Nairn of Blairgowrie the most serious mortality is amongst two-year-olds and three-year-olds. All breeds are apparently susceptible.

Symptoms.—Two forms of the disease may be recognized, namely, **acute** and **subacute.**

The **acute form** is characterized by an obstinate impaction of the large colon. In the earlier stages the animal is dull and may be lying more than usual, evincing some abdominal pain and often dribbling of saliva. Shivering may be observed and sometimes sweating with a patchy distribution. Up to this point the case resembles a mild attack of colic. The temperature and respirations are very little altered, and the former rarely exceeds 102° F. The pulse becomes smaller and quicker than normal. On examining the rectum the mucous membrane feels sticky and a small amount of firm fæces may be found. There is rarely any quantity of fæcal matter in the rectum, but the large colon can be felt containing a firm dryish mass, on which little or no impression can be made with the fingers. About this time some difficulty in swallowing may be observed, and at intervals symptoms of choking are evinced, the chin being drawn into the neck, and a spasm results in a flow of saliva and ingesta through the mouth and nostrils. Twitchings of the muscles occur in the region of the shoulders and the thighs. The acute form runs a rapid course and may be fatal in from three to five days.

In the **subacute form,** or as it is sometimes called, the **chronic type,** the symptoms may commence with colicky pains, but the main characteristic consists of rapid and great emaciation, and the patient may linger for weeks or months. The affected animal eats and drinks sparingly. Spasms of the throat are observed and intermittent regurgitation from the nostrils. The muscular tremors are seen in the shoulders and thighs and profuse soapy perspiration occurs under the tail, and in some cases on the head and the neck under the forelock and mane. The temperature ranges between 100° and 102° F., and the pulse is increased to a varying degree, ranging from 60 to 100 per minute, and is very small and hard. The patient often assumes a quite characteristic posture, the flanks being very much tucked up and the legs all more or less under the body. On examination per rectum the colon is not found impacted in this form. The affected animal rarely lies down, and if he does it is not for long, until the exhaustion has become so extreme that he is unable to rise. Death then occurs in about twenty-four hours.

The writer has met with cases which appear to be the same disease in horses in Essex. The principal symptoms in two of the cases seen in consultation with Mr. Young of Dunmow may serve to indicate the resemblance to the subacute type as described in Scotland.

Case 1.—A bay cart gelding six years old had been ill for three weeks when seen by the writer. He was extremely emaciated, abdomen tucked up, hind legs well under the body. He was occasionally gulping and some greenish material was escaping from nostrils. The pulse was 64, small, but easily felt, temperature 100·5° F. There were marks of sweating in the perineum under the tail, and dried sweat under the forelock and mane. The penis was pendulous, and could be only partially retracted into the sheath. The rectum was empty, but no impaction of the colon could be felt. The case terminated fatally.

Horse suffering from "Grass Disease"—Scotland.

Case 2 was a grey cart gelding, seven years old, on the same farm. This horse had only been ill several days. The temperature was 101° F., pulse 86 and very small. Muscular tremors were very marked in the region of the stifle. Gulping was frequent and liquids were constantly expelled through the nose and mouth. The animal was sweating freely under the tail and less so under the mane and forelock. The mucous membranes were pink and moist, the abdomen tucked up, the hind legs under the body, and the penis half pendulous. This horse was still living two months after the above symptoms were observed by the writer, but he had become very weak and emaciated.

The spread of the disease southward has also been confirmed by G. Elmes of St. Albans, and by J. W. Hall Machcter at Newnham-on-Severn.

Post-mortem Appearances.—In the acute case, the chief feature appears to be met with in the stomach and bowels, the large colon being impacted with firm dry material. The contents of the remainder of the bowel are either liquid or semi-solid. The stomach sometimes is slightly distended and may or may not show inflamed areas. The spleen is somewhat enlarged. In the sub-acute or chronic cases, the post-mortem findings are particularly disappointing considering the duration of the case and the time permitted for the development of lesions. The most marked feature is the emptiness of the intestinal tract, and the corrugation of the mucous membrane somewhat reminiscent of the ileum in cattle in Johne's disease. There is frequently some pneumonia observed.

Treatment.—Up to the present no treatment appears to have been at all successful, and it is impossible yet to suggest any curative measures likely to be effective. Attention should be given, however, to the relief of symptoms in the hope that by ameliorating them the patient may spontaneously overcome the attack. Attention to the diet with nutritious laxative food appears to be more serviceable than medicine. Violent purgatives such as eserine or arecoline appear to be useless. On the assumption that it may be an infection with forage, a complete change should be made, and intestinal disinfectants such as calomel or salol or dimol may be tried. The nervous symptoms may be relieved somewhat by sedatives such as the salicylates and atropine. Cod-liver oil and malt help to maintain the patient's strength.

The use of antitoxic serum has been recommended by Buxton and other supporters of the Botulinus theory, but as a curative measure, up to the present, it has not met with much success. It is probable, however, that even supposing that theory to be correct, the serum may be of small service as a curative agent, but of considerable value as a preventative if injected into horses in infected localities. It is suggested that the toxin produced by the organism resembles in this respect that produced in tetanus, in which it is well known that the preventive effect of the serum is of far greater value than when it is administered after symptoms have been observed. Investigations, however, are still proceeding both with regard to the *Bacillus botulinus* and the *Diplo-streptococcus*, and it would be premature to assume any beneficial result in either case pending the completion of the investigations and the confirmation of the conclusions of the investigators.

Horse suffering from "Grass Disease" or condition simulating it—Essex.

G. H. W.

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